COMPARATIVE EVALUATION OF SERUM C-REACTIVE PROTEIN AND COMPLETE BLOOD COUNT IN CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER NONSURGICAL PERIODONTAL THERAPY

Dissertation submitted to

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment for the degree of

MASTER OF DENTAL SURGERY

BRANCH II

PERIODONTOLOGY

May 2019

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600032

2016 – 2019

CERTIFICATE – I

This is to certify that **Dr. G.KALAIVANI (Reg No: 241613552)** Post Graduate student (2016-2019) in the Department of Periodontology, CSI College of Dental Sciences and Research, has done this dissertation titled **"COMPARATIVE EVALUATION OF SERUM C-REACTIVE PROTEIN AND COMPLETE BLOOD COUNT IN CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER NONSURGICAL PERIODONTAL THERAPY"** under our guidance and supervision in partial fulfillment of the regulations laid down by **The Tamilnadu Dr. M.G.R. Medical University**, Chennai – 600 032 for **M.D.S.,** (Branch –II) **Periodontology** degree examination.

Signature & seal of the HOD

Dr. V.R.BALAJI M.D.S.,
PROFESSOR AND HEAD
Department of Periodontology
CSI College of Dental Sciences and Research
Madurai.

Signature & seal of the principal

Dr.K.THANVIR MOHAMED NIAZI M.D.S.,
PRINCIPAL
CSI College of Dental Sciences and Research
Madurai.

DECLARATION BY THE CANDIDATE

TITLE OF DISSERTATION	**COMPARATIVE EVALUATION OF SERUM C-REACTIVE PROTEIN AND COMPLETE BLOOD COUNT IN CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER NONSURGICAL PERIODONTAL THERAPY**
PLACE OF STUDY	**CSI College of Dental Sciences and Research , Madurai**
DURATION OF COURSE	**3 years**
NAME OF THE GUIDE	**Dr. V.R.BALAJI MDS.,**
HEAD OF THE DEPARTMENT	**Dr. V.R.BALAJI MDS.,**

I hereby declare that no part of the dissertation will be utilized for gaining financial assistance for research or other promotions without obtaining prior permission from the Principal, CSI College of Dental Sciences and Research, Madurai. In addition, I declare that no part of this work will be published either in print or electronic without the prior permission of the guide who has been actively involved in dissertation. The author has the rights to reserve for publishing the work solely with prior permission of the Principal, CSI College of Dental Sciences and Research, Madurai.

Signature & Seal of the HOD **Guide** **Signature of the candidate**

CERTIFICATE II

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI

PLAGIARISM CERTIFICATE

This is to certify the dissertation titled **"COMPARATIVE EVALUATION OF SERUM C-REACTIVE PROTEIN AND COMPLETE BLOOD COUNT IN CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER NONSURGICAL PERIODONTAL THERAPY** "of the candidate **Dr.G.KALAIVANI (Reg No:241613552)** for the award of **MASTER OF DENTAL SURGERY** in the **BRANCH II - PERIODONTOLOGY.**

On verification with the urkund.com website for the purpose of plagiarism check, the uploaded thesis file contains from introduction to conclusion pages and results shows **16 Percentage** of plagiarism in the dissertation.

Date:

Place: Madurai

Signature of the Guide

Dr. G.KALAIVANI
Postgraduate student
Department of Periodontology
CSI College of Dental Sciences and Research
Madurai.

Dr. V.R.BALAJI M.D.S.,
Professor and Head
Department of Periodontology
CSI College of Dental Sciences and Research
Madurai.

ACKNOWLEDGEMENT

ACKNOWLEDGEMENT

First and foremost I thank the **LORD ALMIGHTY** for providing me this opportunity and granting me the capability to proceed successfully.

It is my pleasure to express my deep gratitude to my guide **Dr.V.R.BALAJI M.D.S., Head of the department,** Department of periodontology for his constant support, inspiration and guiding force throughout the course of this study.

I express my sincere appreciation towards **Dr.D.MANIKANDAN M.D.S.,** Reader, Department of periodontology for his guidance, encouragement and valuable insights. His immense knowledge and eye for perfection had a remarkable influence.

I would like to extend my heartfelt gratitude to my co-guide **Dr. G. ROHINI M.D.S.,** Reader, Department of periodontology for her guidance, support, encouragement and immense patience during the preparation of this dissertation and during the course of study.

It gives me great pleasure to thank **Dr. KARTHIKEYAN M.D.S.,** Reader, periodontology who inspired me giving valuable insights during my study.

I thank **Dr. THAMIL SELVAN M.D.S** and **Dr. YAMINI M.D.S** Senior lecturers and **DR. SAHITHYA B.D.S.,** tutor, Department of periodontology for their constant kindness, help and encouragement in conducting this study.

I take this opportunity to express my humble gratitude to **Dr.K.THANVIR MOHAMED NIAZI M.D.S, Principal**, CSI College of Dental Science and Research for his kind permission and encouragement.

My heartfelt appreciation and love to all my colleagues, **Dr. G.ABIRAMI, Dr.A.RAMSUNDAR, JUNIORS, INTERNS AND NON TEACHING STAFFS** for their unyielding support during the period of study.

A special thanks to **ALL THE PATIENTS** who participated in the study. This dissertation would not have been possible without their support and co-operation.

I am infinitely thanks to **MY FAMILY MEMBERS** without whom; I would not have been able to reach this height.

Thank you all.

DR.G.KALAIVANI

CONTENTS

TABLE OF CONTENTS

ABBREVIATIONS

ABBREVIATIONS

1.	AAP	American Academy of Periodontology
2.	ABL	Alveolar bone loss
3.	BC	Basophil Count
4.	BOP	Bleeding on probing
5.	CAL	Clinical attachment level
6.	CBC	Complete Blood Count
7.	CHD	Coronary Heart Disease
8.	CPITN	Community Periodontal Index of Treatment Needs
9.	CRP	C-Reactive Protein
10.	CVD	Cardio Vascular Diseases
11.	DC	Differential Count
12.	EC	Eosinophil Count
13.	ESR	Erythrocyte Sedimentation Rate
14.	GI	Gingival index
15.	Hb	Hemoglobin
16.	HDL	High Density Lipoproteins
17.	hs-CRP	High sensitive C-Reactive Protein
18.	IL-1β	Interleukin-1β

19.	IL-6	Interleukin-6
20.	LC	Lymphocyte count
21.	MC	Monocyte Count
22.	MCH	Mean Corpuscular Hemoglobin
23.	MCHC	Mean Corpuscular Hemoglobin Concentration
24.	MCV	Mean Corpuscular Volume
25.	NCHD	No Coronary Heart Disease
26.	OHI-S	Oral Hygiene Index- Simplified
27.	PBI	Papillary Bleeding Index
28.	PC	Platelet Count
29.	PGE_2	Prostaglandins E_2
30.	PI	Plaque index
31.	PMN	Polymorphonuclear neutrophils
32.	PPD	Periodontal Probing depth
33.	RBC	Red Blood Cell Count
34.	TLC	Total Leukocyte Count
35.	TNF-α	Tumor necrosis factor- α
36.	UNC-15	University of North Carolina Probe-15
37.	vWf	von Willebrand factor

LIST OF FIGURES

LIST OF TABLES

LIST OF GRAPHS

ABSTRACT

ABSTRACT

Aim:

The aim of the present study is to compare and to evaluate the effects of nonsurgical periodontal treatment on serum hs-CRP levels and blood parameters among patients with chronic periodontitis.

Materials and Methods:

A total of 60 patients both males and females in age group of 25-65 yrs were recruited for the study. The study population was divided into three groups: Group I-Healthy controls (n=20), Group II-Patients with moderate periodontitis (n=20) and Group III-Patients with advanced periodontitis (n=20). Blood samples were collected to assess hs-CRP and CBC levels prior and after non surgical periodontal therapy along with periodontal parameters.

Results:

The results were analyzed using t-test and one way ANOVA. The Group II (moderate periodontitis) and Group III (severe periodontitis) showed significant improvement of the clinical and hematological parameters; by statistically significant reduction in hs-CRP level, total leukocyte count, neutrophil count, lymphocyte count, platelet count and ESR level, with statistical significant improvement in total red blood cell count, hemoglobin and MCV values after I month of non surgical periodontal therapy. However, on comparison between two groups there was no significant difference after 1 month.

Conclusions:

Within the limitations of the study it can be concluded that, there is a decrease in CRP levels and hematological parameters after nonsurgical

periodontal treatment, which may possibly prevent the risk of future CVS events.

Keywords:

Periodontitis, hs-CRP, CBC, Non surgical periodontal therapy.

INTRODUCTION

INTRODUCTION

Chronic periodontitis is a disease characterized by an intense inflammatory infiltrate associated with irreversible loss of alveolar bone and connective tissue attachment in the periodontium which ultimately results in the loss of teeth. The mechanism underlying this destructive process involves both direct tissue damage resulting from plaque bacterial products and indirect damage through bacterial induction in the host cells by an activation of inflammatory mediators such as cytokines, prostaglandins and acute phase proteins. In response to bacteria or endotoxins derived from these periodontal pathogens, several acute phase proteins orchestrate the inflammatory cascade to bring about a balance between host and inflammatory stimuli.

One consequence of these inflammatory reactions has been the elevated levels of various acute phase proteins in the serum, plasma, saliva and in gingival crevicular fluid. These acute phase response are initiated by activation of local macrophages and other cells such as fibroblast and endothelial cells, leading to the release of mediators such as Tumor necrosis factor (TNF-α), Interleukin-6 (IL-6) and Interleukin-1β (IL-1β) which in turn stimulate these proteins causing systemic changes.[20]

C-reactive protein (CRP) is a very strong acute phase protein produced in the liver and circulates in the blood. It was discovered by Tillett and Francis laboratory in 1930.[123] It is a systemic marker of inflammation induced by various inflammatory stimuli such as trauma, infection and hypoxia. CRP and other acute phase proteins are usually present at low levels in plasma, but may arise dramatically within 72 hours of injury or with infection.[41] It receives much attention as their level guides decision for diagnosis,

monitoring and therapy for inflammatory process and associated diseases. It was proved to be a significant predictor of future cardiovascular events.

Recent studies have demonstrated a positive correlation between levels of CRP and periodontitis. CRP opsonises bacteria for complement binding and activates complement system when complexed.[41] This may be seen as increased production of CRP due to its activation of inflammatory cytokine production in the presence of bacteria and bacterial products by stimulation of more chemical mediators including interleukin–1 beta (IL-1), tumor necrosis factor alpha (TNF-α), prostaglandins, and matrix metalloproteinases. These products recruit neutrophils to the area and increase the permeability of blood vessels to permit plasma proteins to emigrate into the tissue. As the inflammatory process progresses, other cells such as T-cells, monocytes and platelets are also recruited to the area.

Some studies found that periodontal infection elicits changes in the hematological parameters by exhibiting increase in total leukocyte count (TLC), polymorphonuclear neutrophils (PMN) and lymphocyte count (LC), platelet count (PC) and erythrocyte sedimentation rate (ESR) values.[97,61,45,132] Commonly the total numbers of white blood cells and ESR in peripheral blood have been used as primary diagnostic measure to assess infection and inflammatory status of the disease.[77] Leukocytes are considered as key factors for systemic conditions and diseases, which increased during bacteremia in periodontitis in the systemic circulation. Thus persistent localized infection may have influence over the systemic levels of inflammatory mediators. In order to reduce these inflammatory burdens, the primary goal of nonsurgical periodontal therapy should eliminate these chronic inflammation, arrest periodontal disease progression and should

create an environment conducive to maintenance of health. This nonsurgical therapy found to have various inflammatory changes even in altering C-reactive protein and Complete Blood Count (CBC).[55,49, 60]

Today there is a wealth of cellular and molecular data to find the pathways that the susceptible host to initiate periodontal tissue destruction and to assess the effort of periodontal treatment on the host, but there are few head-to-head comparisons of CRP with CBC evaluation for periodontal disease and its treatment outcome. Measurements of inflammatory markers such as Complete Blood Cell (CBC) count, C-reactive protein (CRP), and Erythrocyte Sedimentation Rate (ESR) suggested for supporting the clinical diagnosis of inflammatory diseases. It is hypothesized that the data will demonstrate the correlation that inflammatory markers with clinical parameters associated with inflammation in periodontitis patients may have variations after nonsurgical periodontal therapy.

The main purpose of this study is to compare and to evaluate the serum level of hs-CRP and CBC in moderate and advanced chronic periodontitis patients before and after nonsurgical periodontal treatment correlated with clinical findings.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

1. To compare the serum levels of **hs-CRP and CBC** in healthy, moderate and advanced generalized chronic periodontitis patient **before** non surgical periodontal therapy.
2. To compare the serum levels of **hs-CRP and CBC** in moderate and advanced generalized chronic periodontitis patient **after** nonsurgical periodontal therapy.
3. To determine the **clinical response** of non surgical periodontal therapy in moderate and advanced chronic periodontitis patients.
4. To assess any **chance of risk** in periodontitis patients by evaluating the serum levels of CRP and CBC.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

❖ *PERIODONTITIS AND ITS EFFECTS ON C-REACTIVE PROTEIN*

Wakai K, Kawamura T, Umemura O, Hara Y, Machida J, Anno T et al (1999)[128] examined a study to determine the possible associations of medical status and physical fitness with periodontal disease. They found older age group with higher C-reactive protein and serum alkaline phosphatase levels and lower high-density lipoprotein associated with periodontal diseases.

Slade, GD, Offenbacher S, Beck, JD, Heiss G and Pankow JS (2000)[117] conducted a study to evaluate association between periodontal disease and CRP levels and to determine whether total tooth loss associated with reduced CRP. The results showed there were associations, showing raised CRP levels with extensive periodontal disease and in edentulous patients.

Wu T, Trevisan M, Genco RJ, Falkner KL, Dorn JP, Sempos CT (2001)[131] examined the relationship between periodontal health and cardiovascular risk factors: serum total and high density lipoprotein cholesterol, C-reactive protein and plasma fibrinogen. The results showed a significant relationship between poor periodontal status and increased C-reactive protein and fibrinogen, concluding these factors may link periodontal disease to elevated cardiovascular risk.

Noack B, Genco RJ, Trevisan M, Grossi S, Zambon JJ, De Nardin E (2001)[89] stated CRP levels were increased in periodontitis patients and found relationship based on severity of periodontal disease and to the periodontal microflora. Subjects

with high levels of mean clinical attachment loss had significantly higher mean CRP levels and more risk for cardiovascular disease

Glurich I, Grossi S, Albini B, Ho A, Shah R, Zeid M, Baumann H et al (2002)[44] focused to determine periodontal disease (PD) as a risk factor for the development of cardiovascular disease (CVD). The data showed increase in hs-CRP and serum amyloid level in periodontitis with or without CVD. This study addressed localized persistent infection may influence systemic levels of inflammatory mediators and have impact on developing inflammation-associated atherosclerotic processes.

Craig RG, Yip JK, So MK, Boylan RJ, Socransky SS and Haffajee AD (2003)[26] investigated the effects of destructive periodontal disease status, severity and progression on the components of the acute phase response. These results suggested that destructive periodontal disease and its progression are associated with the changes in serum components consistent with acute phase response.

Joshipura KJ, Wand HC, Merchant AT and Rimm EB (2004)[59] evaluated the association between periodontal disease and C-reactive protein (CRP), fibrinogen, factor VII, tissue plasminogen activator (t-PA), Low density lipid cholesterol (LDL-C), von Willebrand factor, and soluble tumor necrosis factor receptors 1 and 2. They reported periodontal disease was associated with significantly higher levels of CRP, t-PA and LDL-C with increased risk for developing cardiovascular disease.

Bretz WA, Weyant RJ, Corby PM, Ren D, Weissfel L, Kritchevsk SB et al (2005)[18] studied the levels of systemic markers for inflammation with parameters of periodontal diseases in older people. They found periodontal disease was significantly

associated with higher TNF-α and periodontal pathogens found positive to BANA with significantly higher CRP plasma levels. They conclude periodontal disease and infection may be modifiable risk indicators for elevated levels of systemic inflammatory markers in older people.

Leivadaros E, Van der Velden U, Bizzarro, S, Heggeler, Gerdes VEA, Hoek FJ et al (2005)[73] explored arterial wall thickness and systemic markers C-reactive protein (CRP), fibrinogen, and von Willebrand factor (vWf) associated with atherosclerosis in healthy subjects with and without periodontitis. This study indicates that severe periodontitis found to have higher systemic markers and increased arterial wall thickness when compared to moderate periodontitis, suggesting a link between severe periodontitis and high atherosclerotic risk.

Tuter G, Kurtis B and Serdar M (2007)[125] investigated any relationship between GCF and serum levels of high-sensitivity CRP (hs-CRP) in chronic periodontitis patients (CP) with or without coronary artery disease (CAD). In this study they found serum hs-CRP levels were significantly higher in CP patients than GCF-CRP, concluding there was a correlation between serum hs-CRP levels and clinical parameters and between serum Hs-CRP levels and GCF volume.

Linden GJ, McClean K, Young I, Evans A and Kee F (2008)[74] conducted a study to found an association between periodontitis or tooth loss in a homogeneous group of 60–70-year-old chronic periodontitis patients with CRP levels, measured at two time points at a 10-year interval. They found elevated CRP levels associated with high levels of tooth loss.

Gani DK, Lakshmi D, Krishnan R and Emmadi P (2009)[41] investigated systemic levels of inflammatory markers of cardiovascular diseases like C-reactive protein (CRP) and interleukin-6 in patients with chronic periodontitis, in comparison to periodontally healthy individuals. Periodontitis results in higher systemic levels of CRP and IL-6. These elevated inflammatory factors may increase inflammatory activity in atherosclerotic lesions and potentially increasing the risk for cardiovascular events.

Thakare KS, Deo V and Bhongade ML (2010)[121] evaluated the serum levels of C-reactive protein level (CRP) in periodontitis patients with or without atherosclerosis. Mean serum CRP was significantly higher in periodontitis patients with or without atherosclerosis. These higher serum CRP concentrations with periodontitis add inflammatory burden of the individual and increase the risk of atherosclerosis.

Gomes-Filho, Coelho JM, Cruz S, Pasoos JS, De Freitas, Farias NS, Da Silva RA et al (2011)[46] analyzed the relationship between chronic periodontitis and C-reactive protein (CRP) by considering associated variables in individuals with or without cardiovascular disease. In the chronic periodontitis group, mean CRP levels were found to be higher. This study concluded that even after controlling several confounders, elevated CRP found to be associated with chronic periodontitis patients.

Kanaparthy R, Kanaparthy A, Mahendra M (2012)[63] evaluated serum concentration of CRP can be used as a marker of periodontal disease as well as a risk indicator for cardiovascular disease. Their results indicated an increase in serum CRP

levels in generalized aggressive periodontitis and chronic periodontitis patients and can be used as risk indicator for cardiovascular disease.

Shojaee M, Golpasha MF, Maliji G, Bijani A, Mir SM and Kani SN (2013)[114] observed any comparison between salivary C-reactive protein (CRP) in healthy subjects and patients with periodontal disease. The results showed a significant difference in salivary CRP concentrations between the periodontitis patients and healthy subjects, indicating there is a significant association between periodontitis and salivary CRP concentrations.

Bansal, Dhruvakumar and Pandey (2014)[11] designed a study to compare and evaluate the serum levels of CRP in the healthy gingiva, gingivitis and chronic periodontitis patients. The patients with chronic periodontitis found to have mean hs-CRP levels higher than the patients with gingivitis and with healthy gingiva. Furthermore, the hs-CRP levels increased proportionately with the increasing inflammation.

Podzimek S, Mysak J, Janatova T and Duskova J (2015)[99] designed a study to compare and evaluate the systemic levels of CRP in the peripheral blood samples of patients with gingivitis, chronic and aggressive periodontitis, and gingival recessions and compare them with periodontal clinical parameters. The results indicated that CRP levels increases subsequently with the severity of the periodontal disease and there were positive correlation between CRP levels and bleeding on probing index, when compared to the pocket depth index in both chronic periodontitis and aggressive periodontitis patients.

Deepa D, Gupta C and Gupta A (2016)[35] conducted a cross sectional study for chronic periodontitis patients, with and without CVD. The hs-CRP levels measured and found to be having positive correlation with PPD and GI. Elevated hs-CRP showed an independent association with CVD, incremental to various periodontal measures. These findings suggest that inflammation burden of the individual may added by the periodontitis and thus the risk for cardiovascular events.

Bolla V, Kumari P, Munnangi SR, Sunil Kumar D, Durgabai Y and Koppolu P (2017)[17] compared and evaluated serum C-reactive protein (CRP) levels in subjects with chronic and aggressive periodontitis. They found CRP levels showed a positive correlation with all clinical parameters. Their levels were found to be greater in chronic periodontitis compared to aggressive periodontitis patients with no significance.

Chandy S, Joseph K, Sankaranarayanan A, Issac A, Babu G, Wilson B et al (2017)[22] evaluated serum CRP, fibrinogen in healthy, chronic and aggressive periodontitis patients. The finding of this study showed increased level of serum CRP and plasma fibrinogen with positive correlation found to exist in clinical parameters of chronic periodontitis when compared with healthy controls. Thus it may serve as a diagnostic marker in inflammatory conditions and their levels may serve as biomarker for assessing periodontitis with cardiovascular diseases.

❖ *INFLUENCE OF PERIODONTAL THERAPY ON CRP*

Ebersole JL, Machen RL, Steffen MJ, and Willmann D (1997)[37] conducted a study to detect both C-reactive protein (CRP) and haptoglobin (Hp) in serum of adult periodontitis (AP) patients and normal subjects. Each acute-phase reactant was significantly increased in serum from AP patients with CRP and Hp levels. Their levels showed a significant decrease following scaling and root planing and flurbiprofen administration. These findings indicated that localized infections resulting in increased inflammation and tissue loss in the periodontium elicit systemic host changes manifest by increases in two acute-phase reactants.

Mattila K, Vesanen M, Valtonen c V, Nieminen M, Palosuo T, Rasi V, Asikainen S (2002)[82] compared a quantitative study to evaluate levels of C-reactive protein and fibrinogen level before and after periodontal therapy. They found periodontitis seems to increase C-reactive protein only in some individuals, presumably the ones reacting to it with a systemic inflammatory reaction. Periodontal treatment decreases C-reactive protein levels in these individuals and it may thus decrease their risk of coronary heart disease.

Ide M, McParlin D, Coward M, Lumb P and Wilson RF (2003)[53] designed a study to ascertain if circulating levels of cardiovascular and systemic inflammatory markers could be modified following treatment of periodontal disease. Serum and plasma fibrinogen, CRP, sialic acid, tumor necrosis factor-α and IL-6 were evaluated at baseline, 6 weeks and after 3 months following periodontal therapy. Treatment improved plaque and bleeding scores and reduced probing depth but did not show improvement in levels of systemic markers.

D'Aiuto F, Parkar M, Andreou G, Suvan J, Brett PM, Ready D, Tonetti MS (2004)[31] assessed the degree of response to periodontal therapy associated with any changes in serological markers of systemic inflammation. Periodontal parameters and inflammatory markers C-reactive protein (CRP) and Interleukin-6 (IL-6) were evaluated prior to and 2 and 6 months after delivery of standard non-surgical periodontal therapy. Six months after treatment, significant reductions in serum IL-6 and CRP were observed.

D'Aiuto F, Nibali L, Parkar M, Suvan J, and Tonetti MS (2005)[27] found severe periodontitis has been associated with increased systemic inflammation by estimating the serum inflammatory markers C-reactive protein (CRP), interleukin-6 (IL-6) and cholesterol levels. Two months after periodontal treatment, resulted in significant reductions in serum CRP compared with the untreated control.

Seinost G, Wimmer G, Skerget M, Thaller E, Brodmann M, Gasser R et al (2005)[112] analyzed the markers of systemic inflammation measured at baseline and at follow up. Successful periodontal treatment resulted in a significant improvement compared to baseline, accompanied by a significant decrease in C-reactive protein concentrations. These results indicated that treatment of severe periodontitis reverses endothelial dysfunction.

Yamazaki K, Honda T, Oda T, Ueki-Maruyama K, Nakajima T, Yoshie H et al (2005)[133] assessed the effect of periodontal treatment on the C-reactive protein and proinflammatory cytokine levels in moderate to advanced periodontitis patients. He showed a significant improvement in periodontal parameters with decreased hs-CRP

levels but other proinflammatory cytokine not significantly improved after periodontal therapy.

Etler J, Hinderliter L, Offenbacher S, Beck JD, Caughey M, Brodala N and Madianos PN (2006)[38] conducted a study in otherwise healthy adults with moderate to severe periodontitis to determine if periodontal therapy would result in improved endothelial function and a decrease in serum inflammatory markers. They stated periodontal treatment results in improvements in periodontal pocketing, flow-mediated dilation, and serum IL-6 and serum CRP suggesting an improvement in endothelial function and decreased inflammatory burden.

Tonetti MS, D Aiuto F, **Nibali L, Donald A, Storry C, Parkar M et al (2007)**[124] evaluated severe periodontitis patients after intensive periodontal treatment by comparing endothelial function by measuring the diameter of the brachial artery during flow, and inflammatory biomarkers like CRP,IL-6 and markers of coagulation and endothelial activation before treatment and 1, 7, 30, 60, and 180 days after treatment. After 6 months, the benefits of the oral health showed improvement in endothelial function and decreased inflammatory biomarkers.

Ushida Y, Koshy G, Kawashima Y, Kiji M, Umeda M, Nitta H et al (2008)[126] compared the effect of single-visit full-mouth mechanical debridement (FMD) and quadrant-wise mechanical debridement (QMD) on the levels of serum interleukin (IL)-6, C-reactive protein (CRP) and soluble thrombomodulin levels. They concluded in the quadrant-wise group, serum IL-6 level decreased significantly 1 month after debridement compared with baseline and thrombomodulin levels decreased

significantly in the full-mouth groups but not in the quadrant-wise group. Changes in CRP level were not significant at baseline or after debridement in all the groups.

Nakajima T, Honda T, Domon H, Okui T, Kajita K, Ito H et al (2010)[88] conducted a study to determine periodontal infection alter the serum levels of C-reactive protein (CRP), interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) before and after periodontal treatment. These study results showed periodontal infections have increased serum levels of C-reactive protein (CRP), interleukin-6 (IL-6). After periodontal treatment, their levels decreased, suggesting the risk for CHD.

Freitas CO, Gomes-Filho IS, Naves RC, Cruz SS, Santos CA and Barbosa MD (2011)[40] investigated the effect of periodontal therapy on the levels of CRP levels**.** They showed a statistically significant difference between the CRP levels before and after periodontal treatment. From these findings we can conclude that nonsurgical periodontal therapy showed a tendency to reduce the CRP serum levels.

Kamil W, Al Habashneh R, Khader Y, Al Bayati L, Taani D (2011)[62] performed a study to determine if nonsurgical periodontal therapy has any effect on C-reactive protein **(**CRP) and serum lipid levels in patients with advanced periodontitis. This study demonstrated that nonsurgical periodontal therapy results in a significant reduction in the clinical parameters and serum CRP level but not lipid levels.

George AK and Janam P (2013)[43] conducted a study to determine the effect of nonsurgical periodontal therapy in severe chronic periodontitis patients. They found that after nonsurgical therapy there were decreased in CRP levels and IL-6 levels

when compared to baseline. In this study they concluded that periodontal disease significantly affects the serum levels of systemic inflammatory markers and that nonsurgical therapy could bring about a decrease in the inflammatory burden.

Leite ACE, Carneiro VM and Guimaraes M (2014)[72] investigated the effects of nonsurgical periodontal therapy on levels of high-sensitivity C-reactive protein in the sera and its association with body mass index and high density lipoprotein in subjects with severe periodontitis. They concluded in systemically healthy subjects with periodontitis, periodontal therapy was associated with decreased levels of circulating hs-CRP and increase of high density lipoprotein in serum.

Graziani F, Cei S, Orlandi M et al (2015)[47] conducted a study to compare acute-phase (24-h) and medium-term (3 months) inflammation after quadrant scaling (Q-SRP) versus full mouth scaring (FM-SRP). He concluded that FM-SRP produced a greater acute-phase response after 24 h [threefold increase in C-reactive protein (CRP), twofold increase in interleukin (IL-6), and a slight increase in tumor necrosis factor. But no significant differences in systemic biomarkers were noted between groups at 3 months follow-ups.

Solomon S, Pasarin L, Ursarescu I, Martu I, Bogdan M, Nicolaiciuc O et al (2016)[118] conducted a study to assess the effect of the nonsurgical periodontal therapy on the serum levels of C-reactive protein (CRP) and interleukin-6 (IL-6) in patients with atherosclerosis and periodontal disease. Subjects with an improved periodontal status presented low levels of CRP and IL-6 after 3 months. Thereby a relationship

between the non-surgical periodontal therapy and the systemic parameters were observed.

De Souza AB, Okawa RP, Silva CO and Araujo MG (2017)[34] evaluated serum C-reactive protein (CRP) levels in chronic periodontitis patients and periodontally healthy individuals and assessed the effect of non-surgical periodontal treatment on the CRP levels. After 60 days there were improvement in all periodontal clinical variables and the CRP level decreased significantly in those patients with higher baseline levels of CRP.

Mallapragada S, Kasanal J and Agrawal P (2017)[80] showed the effect of nonsurgical periodontal therapy on circulating serum high sensitivity capsule reactive protein (hs‑CRP) and homocysteine (Hcy) levels in patients with chronic periodontitis before and after 3 months, showing reduction in the mean serum hs-CRP and Hcy concentration.

Aziz AD, Kalekar M, Suryakar DN, **Kale R** , **Benjamin T, Dikshit M (2018)**[10] observed the effect of SRP (scaling and root planning) on Interleukins 6, 10, and C-Reactive Protein (CRP) levels in chronic periodontitis patients with and without diabetes. They concluded that SRP therapy was effective in improving clinical and biochemical markers in the non-diabetic patients compared to diabetic patients with chronic periodontitis.

❖ *PERIODONTITIS AND ITS EFFECTS ON HEMATOLOGICAL PARAMETERS.*

Kweider M, Lowe GD, Murray GD, Kinanei GD (1993)[70] investigated plasma fibrinogen and white blood cell count in healthy and periodontitis patients. The results indicated inflammatory disease may be a determinant of fibrinogen level and white cell count in the general population, and that fibrinogen and white cell count may be two mediators of the link between periodontal disease and myocardial infarction.

Imaki M, Ogawa Y, Yoshiday, Uchida M and Tanada S (1999)[54] investigated the relationship between oral health and the total leukocyte count in the cohort study. The relationship between the total leukocyte count and the oral condition of the subjects classified according to their smoking habits and they were investigated over a 5-year period. Among the current smokers, the total leukocyte count was highest each year among all the groups.

Hutter JW, Van der Velden J, Huffels RAM, Hoek FJ, Loos BG (2001)[51] investigated red cell parameters in periodontitis patients based on severity, with signs of anemia. Overall data analysis indicated that periodontitis patients have a lower hematocrit, lower numbers of erythrocytes, lower hemoglobin levels and higher erythrocyte sedimentation rates.

Monteiro AM, Jardini MA, **Alves, S, Giampaoli, V, Aubi EC, Neto AM, Gidlund M (2009)**[86] conducted a study to investigate the association between chronic periodontitis and cardiovascular risk markers. The levels of leukocyte and neutrophil

counts were significantly higher compared to controls, suggesting association between coronary artery disease and periodontitis.

Aljohani H et al (2010)[3] found association between hemoglobin level and the severity of chronic periodontitis. The correlation between hemoglobin and the means of clinical attachment loss and bleeding on probing was insignificant. The mean hemoglobin found to be insignificantly correlated with the number of missing teeth. No association between hemoglobin levels and periodontal status was found.

Gokhale SR, Sumanth S and Padhye AM (2010)[45] demonstrated a study to show any correlation between signs of anemia and severe chronic periodontitis by assessing red cell parameters. The results showed that patients with chronic periodontitis had lower values of hematocrit, number of erythrocytes, and hemoglobin compared to healthy controls.

Pejcic A, Kesic L, Pesic Z, Mirkovic D and Stojanovic M (2011)[97] conducted study to investigate the relationship between white blood cell counts in subjects with healthy periodontal tissues, moderate and severe periodontitis. The results indicated a significantly higher count of neutrophils, lymphocytes and total leukocytes and proved to exhibit significant relationship between total leukocyte count, neutrophil count and different forms of periodontal disease.

Al-Rasheed A (2012)[4] evaluated a study to estimate the white blood cell (WBC) and platelet counts in chronic periodontitis patients. The results in these study showed that periodontitis patients demonstrated a significantly higher WBC counts than that of

control patients. The platelet count of patients with chronic periodontitis was also significantly higher compared to the healthy group.

Lopez R, Loos BG and Baelum V (2012)[78] assessed periodontitis patients with hemogram findings. Eosinophil counts and mean platelet volumes were increased in number and associated with the parameters of periodontitis and may reflect the roles in periodontal inflammation.

Prakash S, Dhingra K and Priya S (2012)[101] evaluated and compared the red blood cell count, levels of hemoglobin, hematocrit, erythrocyte sedimentation rate (ESR), serum iron and serum ferritin between subjects with and without periodontitis. The results showed ESR level was found to be higher and other values were found to be not significant, concluding that the severity of periodontitis may not affect the hematological and biochemical parameters of an individual.

Khan SI, Iqbal S, Haris KT, Chandramohan S and Senthil Kumar S (2014)[64] investigated the association between hemoglobin level and the severity of chronic periodontitis in systemically healthy male patients. Results showed that patients suffering from chronic periodontitis have lower values of hemoglobin compared to healthy controls. Thus, based on these results it can be concluded chronic periodontitis like any other chronic disease can lead to anemia.

Hegde S, Riyas, Kashyap R, Arun Kumar (2014)[50] compared the red blood parameters in patients with clinically healthy gingiva and chronic periodontitis for signs of anemia. Data analysis showed that patients with chronic periodontitis had lower values of hematocrit, number of erythrocytes, and hemoglobin and increased

ESR level compared to healthy group. No remarkable differences in levels of MCH, MCHC and MCV were found between test and control group.

Kolte RA, Kolte AP and Deshpande NM (2014)[68] compared the various blood parameters in healthy subjects and severe chronic periodontitis patients. The results showed periodontitis group have lower erythrocyte count and mean corpuscular hemoglobin concentration (MCHC), and increased total leukocyte count (TLC) and neutrophil, lymphocyte, and eosinophil count, compared to the healthy control group pointing periodontitis may tend towards anemia and there is marked leukocytosis due to increased number of circulating neutrophils and lymphocytes.

Kumar BP, Khaitan T, Ramaswamy P, Sreenivasulu P, Uday G, Velugubantla RG (2014)[69] evaluated levels of white blood cells (WBCs) and platelets in chronic periodontitis patients. It was found levels of WBC count were higher in patients with chronic periodontitis when compared with controls whereas the platelet count was lower in the case group. These results suggest an increased WBC count have a risk for cardiovascular diseases in periodontitis patients.

Mishra P, Agarwal S, Devraj CG, Nayak PA, Yadav A, Sharma S (2014)[83] aimed at finding the relation between erythrocyte parameters and chronic periodontitis. The results showed the mean values of hemoglobin (Hb) and red blood cell indices were significantly lower, while the value of ESR was significantly higher in test group as compared to control group, suggesting a mild anemia.

Khan NS, Luke R, Soman RR, Krishna PM, Safar IQ and Swaminathan SK (2015)[65] assessed the red blood cell parameters based on the severity of periodontitis.

Their analyzed data showed red blood cell parameters were a significantly decreased in severe form periodontitis.

Suchetha A, Chandran N, Bhat D, Sapna N, Sravani K and Mundinamane DB (2015)[119] conducted a study to evaluate and compare the hematological parameters [Red blood cells (RBC), Hemoglobin (Hb), Erythrocyte sedimentation rate (ESR)] and to assess the relationship between periodontitis and anemia. The mean ESR score was found to be highest and RBC and Hb were found to be lower in periodontitis group, followed by gingivitis group and healthy group. Thus it concludes the positive relationship between hematological parameters and severity of periodontal disease

Vaishali S, Parveen S, Vishunupriya V and Gayathri R (2016)[127] designed a study to compare the white blood cell count in patients with chronic periodontitis with the healthy control. The changes in WBC count in periodontitis were analyzed.

Chandy S, Krishnan V, Anila S, Issac AV (2018)[23] compared peripheral blood levels in healthy subjects, chronic and aggressive periodontitis patients. The results showed an increase in WBC and platelets in chronic and aggressive periodontits groups. RBC and hemoglobin level were found to decrease in both the study groups when compared to healthy controls. This study indicates that there is correlation between periodontitis and peripheral blood levels based on intensity and severity of disease.

❖ *INFLUENCE OF PERIODONTAL THERAPY ON THE HEMATOLOGICAL PARAMETERS.*

Christan C, Dietrich T, Hagewald S, Kage A and Bernimoulin JP (2002)[24] examined the effect of non-surgical therapy on white blood cell in aggressive periodontits patients. After periodontal treatment WBC counts, neutrophil and platelet counts significantly decreased suggesting therapeutic intervention may reduce these inflammatory loads.

Agarwal N, Kumar V, and Gujjari S (2009)[1] estimated periodontal management associated with hematological levels. The results showed that correction of periodontal inflammation resulted in a significant increase in hemoglobin levels and erythrocyte counts. The erythrocyte sedimentation rate showed a reduction indicating resolution of periodontal inflammation. There was a significant, but much lesser, improvement in MCV, MCH and MCHC values.

Taylor B, Tofler G et al (2010)[120] conducted a study to determine whether initial periodontal treatment has a beneficial effect on systemic markers of inflammation and cardiovascular risk. Significant increases in hemoglobin and hematocrit were seen after treatment, showing that initial periodontal treatment, a relatively simple and cost-effective intervention, has systemic effects.

Pradeep AR, Anuj S, and Raju P (2011)[100] investigated whether patients with chronic periodontitis have an anemic status, and analyzed the effect of non-surgical periodontal therapy on the anemic status of subjects over a 6-month period. Results showed more than 33% patients had hemoglobin concentrations below normal and all

red blood cell parameters are significant with clinical parameters after treatment. This study strengthens that chronic periodontitis may lead to anemia and provides evidence that non-surgical periodontal therapy can improve the anemic status of patients with chronic periodontitis with greater improvement in females.

Sambashivah, Rebentish PD, Kulal R, Bilchodmath S (2011)[109] compared and evaluated the effect of one and two stage non-surgical therapy on leukocyte count (TLC) and differential count (DLC) in periodontitis patients. There were significant reductions after periodontal therapy in both groups. These results indicate the systemic effect on blood count in periodontitis patient altered after therapeutic intervention.

Malhotra R, Kapoor A, Grover V, Grover D (2012)[79] evaluated the clinical and hematological parameters to analyze the changes after provision of phase I therapy. The mean values of EC, Hb and HCT were significantly lower in chronic periodontitis, and showed a significantly greater increase at 3 months of observation after periodontal therapy. The values of MCV, MCH and MCHC showed a non significant change during the same observation period.

Banthia R, Jain P (2013)[12] conducted an intervention study to investigate the effect of non-surgical periodontal therapy on total leukocyte count (TLC), differential leukocyte count (DLC) and platelet count in patients with chronic periodontitis. There were statistically significant decrease in TLC, DLC, platelet count at baseline and two weeks following phase I therapy, thereby focusing the importance of periodontal

therapy to reduce the TLC and platelet count and possibly decreasing the risk for the development of cardiovascular disease.

Patel MD, Shakir QJ, and Shetty A (2014)[94] observed the effects of non-surgical periodontal therapy in systemically healthy male patients having chronic periodontitis. The results showed an improvement in both clinical and red blood cell parameters from baseline to 6 months after non-surgical periodontal therapy.

Jain K, Das SJ, Dwivedi S, Jain R, Nugala B and Jain M (2016)[57] carried out study to evaluate the effect of periodontal treatment on levels of red blood cell parameters related to anemia of chronic disease in patients with chronic periodontitis. After 9 months there were improvements in clinical parameters and increase in levels of red blood cells, hemoglobin concentration and packed cell volume together with decrease in values of ESR when compared to baseline.

Siddeshappa ST, Nagdeve S, Yeltiwar RK, Parvez H, Deonani S, Diwan V (2016)[115] evaluated the effect of nonsurgical periodontal therapy on clinical and hematological parameters like total leukocyte count (TLC), differential leukocyte count (DC), platelet count and erythrocyte sedimentation rate (ESR) were assessed at 1 week and 2 weeks in periodontitis patients. In this study it had been concluded that there were significant results showing decrease in clinical and hematological parameters after nonsurgical periodontal therapy.

Kahn S, Imperial RC, Menezes CC, Dias AT, Rodrigues WJ, Barceleiro M and Moreira P (2017)[60] demonstrated the study to assess the effects of non-surgical periodontal treatment on the complete blood count, glycemic and lipid profiles in

cardiopathic patients with indication for surgical revascularization. Their results showed significant reduction in probing depth and reduction in total cholesterol and high density lipoprotein. The differences in clinical attachment level and values of leukocyte count, glucose, hemocyte and hemoglobin were not statistically significant.

Kalsi DS, Sood A, Mundi S and Sharma V (2017)[61] designed a study to assess the effect of blood parameters like ESR, TLC, PMN count, lymphocyte count DLC, Hb, BT and their periodontal status after 21 days of nonsurgical periodontal therapy. There were significant reductions in counts of PMNs, lymphocyte count, ESR, TLC, DLC and BT and non significant results in Hb after non surgical periodontal therapy.

Sangeetha S, Devi R, Nalini E and Prasad AK (2017)[110] investigated the effect of nonsurgical periodontal therapy on various hematological parameters (TLC, DC, ESR and platelet count) in patients with chronic periodontitis. There were significant reductions in the TLC and ESR values, but no significant reductions in DC and platelet count observed at 2 weeks intervals after non surgical periodontal therapy.

Parihar S, Kumar N, Bhatnagar A, Parihar AV (2018)[93] evaluated the hematological parameters obtained from systemically healthy male patients with chronic periodontitis measuring Hemoglobin level (Hb), Erythrocyte Count, Erythrocyte Sedimentation Rate (ESR), Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH) and Mean Corpuscular Hemoglobin Concentration (MCHC) with clinical parameters. After non surgical periodontal therapy it leads to an improvement in these hematological and clinical parameters.

❖ *PERIODONTITIS AND ITS EFFECT ON CRP AND HEMATOLOGICAL PARAMETERS*

Fredriksson MI, Figueredo CMS, Gustafsson A, Bergstrom KG and Asman BE (1999)[39] compared the systemic effects of periodontitis and smoking to study the peripheral neutrophils hyperreactivity. The periodontitis patients had higher concentrations of C-reactive protein (CRP), neutrophil count, haptoglobin, and α-1 antitrypsin levels influenced by cigarette smoking. The effects of periodontitis with smoking have more impact on CRP and neutrophils.

Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden U (2000)[76] found higher median CRP level, IL-6 and Leukocyte levels in generalized and localized chronic periodontitis patients than in controls. These finding demonstrated that higher numbers of neutrophils, IL-6 and CRP levels were correlated with each other, and on the severity of disease progression.

Persson GR, Pettersson T, Ohlsson O, Renvert S (2005)[98] assessed periodontal status, leukocyte count and hs-CRP serum levels. The results concluded elevated serum hsCRP concentration and serum WBC counts associated with acute coronary heart disease and also elevated serum hsCRP values associated with periodontitis in subjects with no evidence of CVD.

Buhlin K, Gustafsson A, Pockley AG, Frostegard J and Klinge B (2009)[19] compared complete blood counts, levels of high density lipoproteins (HDL), total cholesterol, haptoglobin, elastase, C-reactive protein (CRP), IL-6, TNF alpha receptor-1, alpha-1-antitrypsin and antibodies against human heat shock protein

(Hsp), mycobacterial Hsp and ox LDL. Monocyte counts were elevated and CRP levels were higher in patients, but other inflammatory markers and lipid profile were found to significantly lower in periodontitis patients. These results may provide more chance of association between periodontitis and cardiovascular disease.

Rai B, Kaur J, Kharb S, Jain R, Anand SC and Singh J (2009)[103] analyzed CRP levels, hemoglobin, red blood cell count (RBC), leukocyte cell count and platelet count in periodontitis patient. Total WBC, neutrophil and platelet counts and CRP levels were raised significantly while RBC count and hemoglobin were significantly lowered in periodontitis as compared to controls, suggesting routine screening for CRP in periodontitis patients might be an important tool to prevent heart diseases.

Anitha G, Nagaraj M and Jayashree A (2013)[7] investigated the levels of CRP and PNM cells as a marker of inflammatory host response in the serum of chronic periodontitis patients and in patients with CVD. On comparison, OHI-S Index, GI, mean PD, CRP and PMN values showed significant difference in chronic periodontitis patients with CVD than chronic periodontitis patients without CVD.

Ana P, Draginja K, Dimitrije M, Ivan M and Mariola S (2013)[6] conducted a study to find any relationship between periodontitis and systemic inflammatory markers, as well as, if there is a relation to severity of periodontitis. The leukocyte count, CRP, and fibrinogen were assed. The results showed increased levels CRP and fibrinogen in periodontitis patients. This study suggests the marker levels in periodontitis patients depend of severity of the disease and increase in the WBC, CRP and fibrinogen levels appears to be contributing factors for CVD.

❖ *INFLUENCE OF PERIODONTAL THERAPY ON C-RECATIVE PROTEIN AND HEMATOLOGICAL PARAMETERS.*

D'Aiuto F, Nibali L, Parkar M and Tonetti MS (2005)[30] conducted a clinical trial with the objective of developing a novel in vivo inflammatory model. Subjects suffering from severe periodontitis, after periodontal therapy showed decrease in TNF-alpha, IL-6, CRP and Fibrinogen levels and differential blood counts measured at 7 and 30 days.

Montebugnoli L, Servidio D, Miaton RA, Prati C, Tricoci P, Melloni C et al (2005)[85] conducted a study to assess whether periodontal treatment cause a change in systemic inflammatory [C-reactive protein (CRP), leukocyte**s**, fibrinogen] and haemostatic factors [von Willebrand factor, fibrin D-dimer and oxidized-low density lipoprotein (Ox-LDL)]. All systemic inflammatory markers were found to decreased, providing an evidence altering periodontal status may influence the systemic inflammatory and haemostatic situation.

Taylor BA, Tofler GH, Carey HMR, Morel-Kopp MC, Philcox S, Carter TR et al (2006)[120] conducted a study to found any relationship between periodontitis and systemic inflammatory markers in a treatment intervention model. This study shows that elimination of advanced periodontitis by full-mouth tooth extraction reduces C-reactive protein (CRP), plasminogen activator inhibitor-1 and fibrinogen, and white cell, platelet counts and thrombotic markers of cardiovascular risk supporting the hypothesis that treatment of periodontal disease may lower cardiovascular risk.

Bokhari SA and Khan AA (2009)[16] evaluated systemic levels of inflammatory markers (CRP, fibrinogen, and WBC counts) prior to and 1 month after non surgical periodontal therapy. In all subjects, CRP, fibrinogen, and WBC counts were reduced significantly after periodontal therapy. Thus periodontal treatment resulted in significant decreases in BOP and PD and lowered serum inflammatory markers in patients with CHD or NCHD by decreasing the risk.

Marcaccini M, Meschiari CA, Zuardi LR, de Sousa TS, Taba M , Teofilo JM et al (2009)[81] evaluated whether non-surgical periodontal therapy reduce hematological parameters (CBC) and inflammatory disease markers interleukin (IL)-6, high-sensitivity C-reactive protein (hs-CRP), CD40 ligand, monocyte chemoattractant protein (MCP)-1, soluble P-selectin (P-selectin), soluble vascular adhesion molecule (VCAM)-1, and soluble intercellular adhesion molecule (ICAM)-1. After 3 months, all hematological and inflammatory markers were reduced and decreased the risk for cardiovascular events.

Graziani F, Cei S, Tonetti M, Paolantonio M, Serio R, Sammartino G, Gabriele M and D'Aiuto F (2010)[47] compared leukocyte counts, C-reactive protein (CRP), serum amyloid-A (SAA) and D-dimers and renal function. Patients undergoing periodontal treatment experience reduction of systemic inflammation of a greater magnitude after non-surgical than surgical periodontal therapy.

Gani DK, Mallineni SK, Ambalavanan, Ramakrishnan, Deepalakshmi and Emmadi P (2012)[42] investigated C-reactive protein (CRP), interleukin-6 (IL-6), total leukocyte count and differential count in patients with generalized and localized

chronic periodontitis, in comparison to healthy individuals. The results showed C-reactive protein level, IL-6 in generalized and localized periodontitis groups were statistically significant when compared to healthy patients. Total leukocyte count, neutrophil count and neutrophil percentage were also statistically significant in generalized periodontitis, when compared to localized and healthy groups.

Pabolu CM, Mutthineni AB, Chintala S and Mutthineni NN (2013)[92] compared the effect of one stage versus two stage non surgical periodontal therapy on leukocyte count and C reactive protein**.** The results pointed out that there were significant differences on blood counts in periodontitis patients treated either by one stage or full mouth disinfection.

Zhou SY, Duan XQ, Hu R, Ouyang XY (2013)[134] assessed change in serum levels of C-reactive protein (CRP), tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6) , lipid profile markers and white blood cell count. The results showed non-surgical periodontal therapy decreased serum CRP, TNF-α and IL-6 levels in chronic periodontitis subjects with stable CHD, which could help to reduce the inflammatory burden of stable coronary heart disease subjects.

Musalaiah S, Anupama M, Nagasree M, Krishna M, KumarA, and Kumar PM (2014)[87] compared the efficacy of nonsurgical periodontal therapy on red blood cell (RBC) parameters and hs‐CRP in chronic periodontitis patients with anemia. This study highlighted that chronic periodontitis may lead to anemia and provides evidence that nonsurgical periodontal therapy can improve the anemic status and reduce levels of hs‐CRP in patients with chronic periodontitis.

Barot and Chandran (2015)[13] determined the effect of nonsurgical periodontal therapy on serum CRP, WBC and OHI-S level in patients with aggressive and chronic periodontitis. After nonsurgical periodontal therapy, mean CRP levels and WBC were found to decrease after one month. There were statically no significant changes in levels of serum CRP in aggressive and chronic periodontitis. Both forms of periodontitis are associated with increased systemic inflammatory response with severity of disease progression, but improvement in periodontal health did not influence the levels of serum CRP.

Hada DS, Garg S, Ramteke GB and Ratre MS (2015)[48] evaluated the effect of nonsurgical periodontal treatment (NSPT) on the cardiovascular clinical parameters like pulse, respiratory rate, blood pressure (BP) and biochemical parameters like high sensitive C-reactive protein (hs-CRP), lipid profile, and WBC count; at baseline, 1, 3, and 6 months of CHD patients. No significant results were obtained in these parameters after effective non surgical periodontal therapy.

Ramich T, Asendorf A, Nickles K, Oremek GM, Schubert, Nibali L et al (2018)[105] assessed the long-term effect of active periodontal therapy on serum inflammatory parameters in patients with aggressive (AgP) and chronic (ChP) periodontitis in a non-randomized clinical study. Neutrophil elastase (NE), C-reactive protein (CRP), lipopolysacchrides binding protein, interleukin 6, 8, and leukocyte counts were assessed at baseline, 12 weeks and 60 months.NE and CRP were significantly higher in AgP than ChP at baseline and 60 months. For leukocyte counts in ChP, significant changes were observed despite comprehensive periodontal treatment.

MATERIALS AND METHODS

MATERIALS AND METHODS:

The present study was conducted in 60 subjects; randomly selected between age group of 25-65 yrs, irrespective of their sex. Subjects were selected among the patients who visited the department of periodontology in CSI College of dental science and research for their routine periodontal treatment needs. They were screened and recruited for this study.

Institutional research and ethical committee approval was obtained prior to the start of study [CSICDSR/IEC/0027/2016]. Written consent in vernacular language was obtained from the subjects in the study.

The venous blood was collected from the patient for evaluating serum CRP and CBC level before treatment and one month after non surgical periodontal treatment. Clinical periodontal examination was also done by using standard periodontal probe University of North Carolina Probe (UNC-15) before treatment and one month after treatment.

SUBJECT GROUPS:

The study population was divided into three groups.

Group 1	**Control - Healthy Periodontium**	**20 Patients**
Group 2	**Test group - Moderate periodontitis**	**20 Patients**
Group 3	**Test group- Advanced periodontitis**	**20 Patients**

Group 1: comprise patients with clinically healthy periodontium with no evidence of attachment loss or pockets greater than 3 mm.[20]

Based on American Academy of Periodontology (AAP) Task force report 2015 on the update to the 1999 classification of periodontal diseases and conditions group 2 and group 3 were selected on the basis of severity of periodontitis.[5]

Group 2: comprise patients with moderate periodontitis having PD ≥5mm and <7mm in two or more interproximal sites, presence of bleeding on probing, with radiographic bone loss 16% to 30% root length or >3 mm≤ 5 mm may be observed, with grade I furcation involvement and CAL 3-4 mm.

Group 3: comprise patients with advanced periodontitis with the presence ≥7 mm PD, presence of bleeding on probing, mobility, with radiographic bone loss >30% root length or >5 mm may be observed, with >grade I furcation involvement and >5mm of CAL.

INCLUSION CRITERIA:

- Age 25-65 years
- Minimum 20 teeth
- Apparently healthy patients
- Periodontal probing depth >5mm.
- Radiographic evidence of bone loss.

EXCLUSION CRITERIA:

- Patient with antibiotic therapy for past 6 months
- Smokers
- Pregnancy and lactating

- Patient with any systemic diseases and chronic illness
- Patient who had undergone periodontal treatment in the past 6 months.

CLINICAL PARAMETERS:

1. Plaque index(PI) (Silness and Loe)
2. Gingival index (Loe and Silness)
3. Papillary Bleeding Index (H.R. Muhlemann and S.Son)
4. Probing Pocket Depth (PPD)
5. Clinical attachment level (CAL)
6. Periodontal index (A.L.Russell)

PLAQUE INDEX (PI) [Silness P and Loe H- 1961]:

Plaque index is unique among the indices used for the assessment of plaque because it assesses thickness of plaque at the gingival area of the tooth. The full mouth examination has been done by assessing four gingival areas of the tooth, the disto-facial, facial, mesio-facial, and lingual/palatal surfaces. The indices for each of the teeth are added and then divided by the total number of teeth examined. The scores range from 0 to 3.

SCORING CRITERIA FOR THE PLAQUE INDEX:

SCORE	CRITERIA
0	No Plaque
1	Separate flecks of plaque at cervical margin of the tooth
2	A thin continuous band of plaque at the cervical margin.

3	A band of plaque wider than 1 mm but covering less than 1/3 of the crown of the tooth.
4	Plaque covering at least 1/3 but less than 2/3 of the crown
5	Plaque covering 2/3 or more of the crown

GINGIVAL INDEX (GI) [Loe H and Silness J-1963]:

This index is used to assess the severity of gingivitis based on color, consistency, and bleeding on probing. The full mouth examination has been done by assessing **four gingival areas of the tooth, the disto-facial, facial, mesio-facial, and lingual/palatal surfaces.** The indices for each of the teeth are added and then divided by the total number of teeth examined.

SCORING CRITERIA FOR THE GINGIVAL INDEX:

SCORE	CRITERIA
0	Normal Gingiva
1	Mild Inflammation, Slight change in color, Slight edema, no bleeding on probing.
2	Moderate Inflammation, redness, edema, glazing, bleeding on probing.

3	Severe Inflammation, marked redness and edema, ulceration, tendency to spontaneous bleeding.

PAPILLARY BLEEDING INDEX (PBI) [H.R. Muhlemann and S.Son -1977]:

It is used for assessment of gingival bleeding. A probe is carefully inserted into the gingival sulcus at the base of the papilla on the mesial aspect, and then moved coronally on the distal aspect of the same papilla recording the intensity of bleeding.

SCORING CRITERIA FOR THE PAPILLARY BLEEDING INDEX:

SCORE	CRITERIA
0	No Bleeding
1	A single discreet bleeding points appears
2	Several isolated bleeding points or a single line of blood appears
3	The interdental triangle fills with blood shortly after probing
4	Profuse bleeding occurs after probing, blood flows immediately into the marginal sulcus.

PERIODONTAL PROBING DEPTH (PPD):

Periodontal probing depth was measured as the distance in millimeters (mm) from the free gingival margin to the bottom of the pocket. It was measured with a **standard periodontal probe (UNC-15)**. The periodontal probe should be inserted parallel to the vertical axis of the tooth and "walked" circumferentially. **Each tooth was scored for six sites: disto-facial, mid-facial, mesio-facial and disto-lingual, mid-lingual, mesio-lingual surfaces.** Probing pocket depth score for a tooth was obtained by adding six values per tooth and dividing by six. Scores of each tooth are added and then divided by number of teeth examined to give pocket depth score for the individual.

CLINICAL ATTACHMENT LEVEL (CAL):

The level of attachment is the distance between the base of the pocket and a fixed point on the crown such as cementoenamel junction (CEJ). The clinical attachment level was measured using periodontal probe (UNC -15).**Each tooth was scored for six sites: disto-facial, mid-facial, mesio-facial and disto-lingual, mid-lingual, mesio-lingual surfaces.** CAL score for a tooth was obtained by adding six values per tooth and dividing by six. Scores of each tooth are added and then divided by number of teeth examined to give CAL score for the individual.

PERIODONTAL INDEX [A.L.Russell, 1956]:

It is used for assessing and scoring the periodontal status of the population. A Mouth mirror and probe is used for examination. The full mouth examination was done. The indices for each of the teeth are added and then divided by the total number of teeth examined.

Scoring Criteria for periodontal Index:

SCORE	CRITERIA
0	**Negative:** There is neither overt inflammation in the investing tissues nor loss of function due to destruction of supporting tissue. Radiographic features are normal.
1	**Mild gingivitis**: There is an overt area of inflammation in the free gingiva, which does not circumscribe the tooth.
2	**Gingivitis**: Inflammation completely circumscribes the tooth but there is no apparent break in the epithelial attachment.
4	Used only when radiographs were available.
6	**Gingivitis with pocket formation**: The epithelial attachment has been broken and there is a pocket. There is no interference with normal masticatory function; the tooth is firm in its socket and has not drifted. Radiographic features suggest there is horizontal bone loss involving the entire alveolar crest up to half of the length of the root.
8	**Advanced destruction with loss of masticatory function:** The tooth may be loose; may have drifted; may sound dull on percussion with a metallic instrument; may be depressible in its socket. Radiographic features suggest advanced bone loss involving more than half of the tooth root or a definite infrabony pocket with widening of periodontal ligament. There may be root resorption or rarefaction at the apex.

SEROLOGICAL AND HEMATOLOGICAL PARAMETERS:

- **High sensitive C- Reactive Protein** (hs–CRP)- mg/L
- **Complete Blood Count** (CBC) - it includes

HEMATOLOGICAL PARAMETERS		
Total leukocyte count	RBC count	Platelet count
Neutrophil count	Hb	ESR
Lymphocyte count	MCV	
Monocytes count	MCH	
Basophil count	MCHC	
Eosinophil count	Hemocrit	

METHOD OF COLLECTION OF BLOOD SAMPLE:

- A total amount of **5 ml** of peripheral blood was collected from the patient by venipuncture in the antecubetal fossa under aseptic condition. Then the sample of 2.5 ml each was collected in two sterile tubes, one tube with an anticoagulant EDTA (ethylenediaminetetraacetic acid) for complete blood count evaluation and other silica coated tube with clotting factor for evaluating CRP.
- The sample was taken at same time interval for all patients.
- It was collected before clinical examination.
- After collecting sample it was sent to laboratory for evaluating serum hs-CRP level and CBC.

hs-CRP AND CBC EVALUATION:

- The high sensitive CRP (**hs-CRP**) assay is done based on an automated **immunonephelometry method**.
- For **CBC** evaluation automated **hemogram counter** is analyzed.
- For **ESR** test, the readings are evaluated based on **Westergrens method.**

TREATMENT PROTOCOL:

- For **group 2 and group 3**, complete supragingival scaling was performed using ultrasonic scalers and subgingival scaling and root planing were performed using Gracey curettes in two visits within 24 hr.
- The patients were given proper oral hygiene with the instructions to use 0.2% of Chlorhexidine digluconate mouthwash twice a day as a supportive oral hygiene measures.
- Patients were advised to report **after I month** for review and for blood sample collection to evaluate hs-CRP and CBC in order to compare it with baseline.

ARMAMENTARIUM

1. Disposable gloves.
2. Disposable mouth mask.
3. Mouth mirror.
4. Graduated William's periodontal probe.
5. UNC –15 color coded probe.
6. Explorer.
7. Tweezer.
8. Ultrasonic scalers.
9. Gracey curettes.
10. Sterilized gauzes.
11. Chlorhexidine mouthwash.
12. Collecting tubes.
13. Disposable needle and syringe.
14. Cotton.
15. Spirit.
16. Tourniquet.
17. hs-CRP reagent kit.
18. CBC auto analyzer.
19. Glass tube for ESR evaluation.

FIGURE 1: ARMAMENTARIUM

FOR CLINICAL PROCEDURE

FOR BLOOD SAMPLE COLLECTION

FIGURE 2: ARMAMENTARIUM FOR SEROLOGICAL AND HEMATOLOGICAL EVALUATION

hs-CRP EVALUATION

hs-CRP analyzer

hs-CRP reagent kit

CBC EVALUATION

Hemogram count auto analyzer

ESR EVALUATION

Westergrens method

FIGURE - 3
CLINICAL PROCEDURES

Blood sample collection and Intervention NSPT for test groups [Group II and group III]

Group I
Healthy periodontium

Group II
Moderate periodontitis

Group III
Advanced periodontitis

Pre operative

No intervention

Intervention- ***NSPT***

After 1 month

Post operative

Post operative

Post operative

STATISTICAL ANALYSIS

STATISTICAL ANALYSIS

Statistical analysis was performed using the SPSS version 21 for data analysis. All the obtained data were presented as mean ± standard deviation. Changes in serum hs-CRP level and complete blood count following periodontal therapy were used as primary outcome variables. A paired t test was used to compare the results obtained within the two groups. ANOVA was used compare between the three groups. p values < 0.05 were considered to indicate significance.

RESULTS

RESULTS

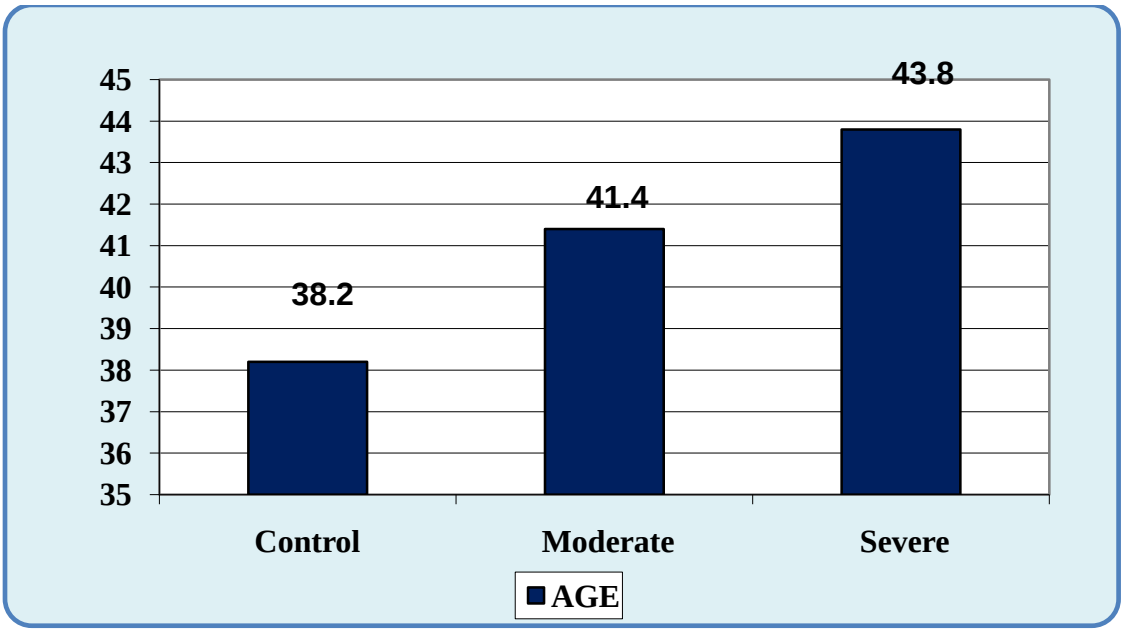
TABLE 1: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO AGE AND GENDER:

GROUPS	AGE (N %)		GENDER	
	<40 yrs	>40 yrs	MALE	FEMALE
GROUP I	13 (65%)	7 (35%)	7 (35%)	13 (35%)
GROUP II	11 (55%)	9(44%)	7 (35%)	13 (35%)
GROUP III	8 (40%)	12 (60%)	11 (55%)	9 (55%)

The study consisted of healthy control (n=20), generalized moderate periodontitis group II (n=20) and generalized advanced periodontitis group III (n=20). As shown in table 1, group I, group II and group III consists of 65 %, 55% and 40% of subjects under mean age of 40 yrs and 35%, 44% and 60% above mean age of 40 yrs. Group II and Group III had more subjects in age group >40 yrs.

Group I and group II consists of 7 males (65%) and 13 females (35%). Group III consists of 11 males (55%) and 9 females (45%). In group I and group II, the subjects were predominantly female as compared to group III. Graph 1, 2 showed the graphical representation of mean age and gender distribution of patient studied.

Graph 1: Graphical representation of mean age



Graph 2: Graphical representation of gender distribution

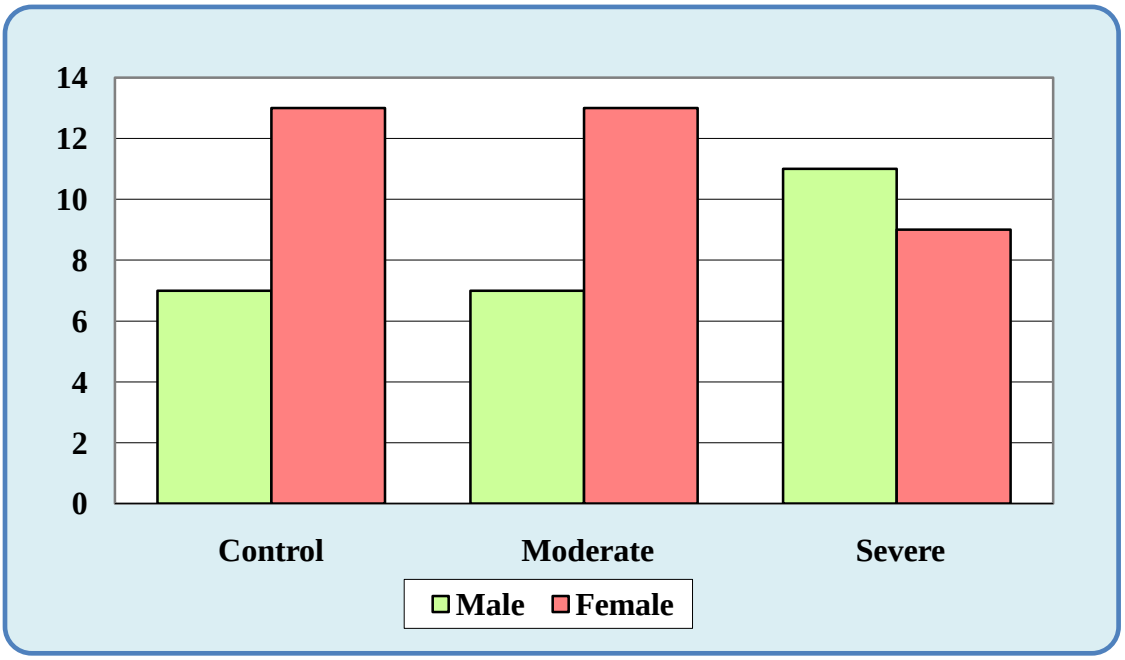


TABLE 2: COMPARISON OF MEAN AND STANDARD DEVIATION (SD) OF PERIODONTAL PARAMETERS AMONG CONTROL (GROUP I) AND TESTS GROUPS (GROUP II AND GROUP III) AT BASELINE AND I MONTH:

PERIODONTAL PARAMETERS	MEAN±SD BASELINE				MEAN±SD I MONTH		
	Group I	Group II	Group III	p value	Group II	Group III	p value
PLAQUE INDEX	0.65± 0.48	1.936± 0.49	2.213± 0.65	<0.001*	0.928 ± 0.66	0.741 ± 0.28	0.253
GINGIVAL INDEX	0.2± 0.41	2.183± 0.449	2.344± 0.406	<0.001*	1.116 ± 0.412	1.067 ± 0.35	0.687
BLEEDING INDEX	0.2± 0.41	2.915± 0.298	3.329± 0.485	<0.001*	1.439± 0.59	1.324 ± 0.49	0.51
PROBING DEPTH	2.45± 0.386	5.909± 0.251	6.58± 0.447	<0.001*	3.704± 0.59	3.368 ± 0.50	0.29
CLINICAL ATTACHMENT LEVEL	2.45± 0.386	.585± 0.35	8.025± 0.792	<0.001*	4.685± 0.58	5.045 ± 1.11	0.917

*** - p value is significant**

The above table 2 showed mean and standard deviation value of PI in group I, group II and group III at baseline is 0.65±0.48, 1.936±0.49 and 2.213±0.65 respectively with the significant difference ($p<0.001$).

The mean and standard deviation value of GI in group I, group II and group III at baseline is 0.2±0.41, 2.183±0.449 and 2.344±0.406 respectively with the significant difference ($p <0.001$).

There is a significant difference (p value <0.001) in PBI at baseline with the mean and standard deviation of 0.2±0.41, 2.915±0.298 and 3.329±0.485 respectively in group I, group II and group III.

The mean and standard deviation of probing depth in group I, group II and group III showed 2.45±0.386, 5.909±0.251 and 6.58±0.447 respectively with p <0.005 showing significant differences among these group.

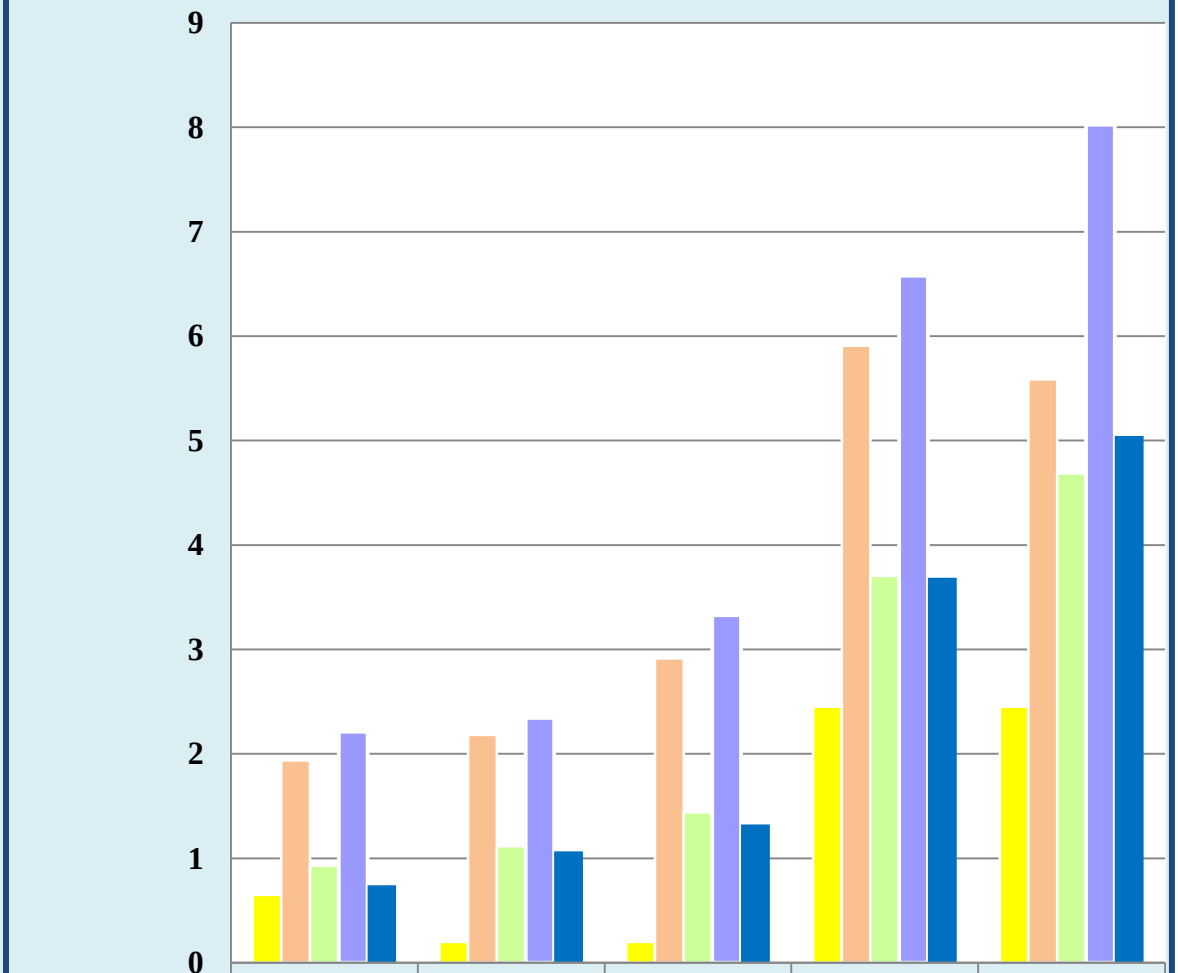
The mean and standard deviation of clinical attachment level (CAL) in group I, group II and group III at baseline showed 0.29±0.456, 5.585±0.35 and 8.025±0.792 respectively with significant difference (p<0.005).

The above table also showed comparison of mean and standard deviation of periodontal parameters after 1 month of nonsurgical periodontal therapy in group II and group III.

There was significant improvement in all periodontal parameters in group II and group III. But the difference among the group II and group III was not statistically significant.

Graph 3 showed the graphical representation of the periodontal parameters in group I, group II and group III at baseline and I month.

Graph 3: Comparison of periodontal parameters in group I, group II and group III at baseline and I month



	PLAQUE INDEX	GINGIVAL INDEX	BLEEDING INDEX	PPD	CAL
Control	0.65	0.2	0.2	2.45	2.45
Mod Preop	1.936	2.183	2.915	5.909	5.585
Post op	0.928	1.116	1.439	3.704	4.685
Sev.preop	2.213	2.344	3.329	6.58	8.025
Post op	0.741	1.067	1.324	3.686	5.045

TABLE 3: COMPARISON OF MEAN AND STANDARD DEVIATION (SD) OF PERIODONTAL PARAMETERS WITHIN GROUP II AND GROUP III AT BASELINE AND AFTER 1 MONTH:

GROUPS		PERIODONTAL PARAMETERS(MEAN±SD)					P VALUE
		PI	GI	BI	PD	CAL	
GROUP II	BASELINE	1.936± 0.49	2.183± 0.44	2.915± 0.29	5.909± 0.251	5.585± 0.35	<0.001*
	I MONTH	0.928± 0.66	1.116± 0.412	1.439± 0.59	3.704± 0.59	4.685± 0.58	
GROUP III	BASELINE	2.213± 0.74	2.344± 0.40	3.329± 0.48	6.58± 0.44	8.025± 0.79	<0.001*
	I MONTH	0.74± 0.28	1.067± 0.35	1.324± 0.49	4.368± 0.50	5.045±1 .11	

*** - p value is significant**

The table 3 showed changes in mean and standard deviation of the periodontal parameters before and after nonsurgical. The mean change in PI before and after intervention was 0.734 in group II and 1.472 in group III, both were satisfactory significant (p <0.001). The mean change in GI before and after intervention was 1.067 in group II and 1.277 in group III and the difference was significant (p <0.001). The mean change in BI before and after intervention was 1.476 in group II and 2.005 in group III, which was significant (p value <0.001). The mean change in PD and CAL at baseline and after 1 month was 2.209 and 0.9 in group II respectively. The mean change in PD and CAL at baseline and after 1 month was 2.212, 2.98 in group III respectively. **The difference among group II and group III was statistically significant (p <0.001).**

TABLE 4: COMPARISON OF MEAN AND STANDARD DEVIATION (SD) OF hs-CRP AMONG CONTROL (GROUP I) AND TESTS GROUPS (GROUP II AND GROUP III) AT BASELINE AND I MONTH:

	BASELINE (MEAN±SD)				I MONTH (MEAN±SD)		
	Group I	Group II	Group III	p value	Group II	Group III	p value
hs-CRP	1.555± 0.953	4.014± 2.92	5.699± 6.505	0.001*	2.715±2. 35	4.328± 4.479	0.917

*** - p value is significant**

The above table 4 showed mean and standard deviation value of hs-CRP in group I, group II and group III was 1.555±0.953, 4.014±2.92 and 5.699 ±6.505 respectively. In group II and group III the hs-CRP were comparatively higher when compared to group I. **On comparing group I, group II and group III, the hs-CRP values were statistically significant (p <0.001).**

This table also showed the comparison of mean and standard deviation of hs-CRP after 1 month of nonsurgical periodontal therapy in group II and group III. **The mean change in the hs-CRP levels at 1 month among group II and group III was not statistically significant.**

Graph 4 showed the graphical representation of hs-CRP in group I, group II and group III at baseline and I month.

Graph 4: Comparison of hs-CRP in at baseline and I month

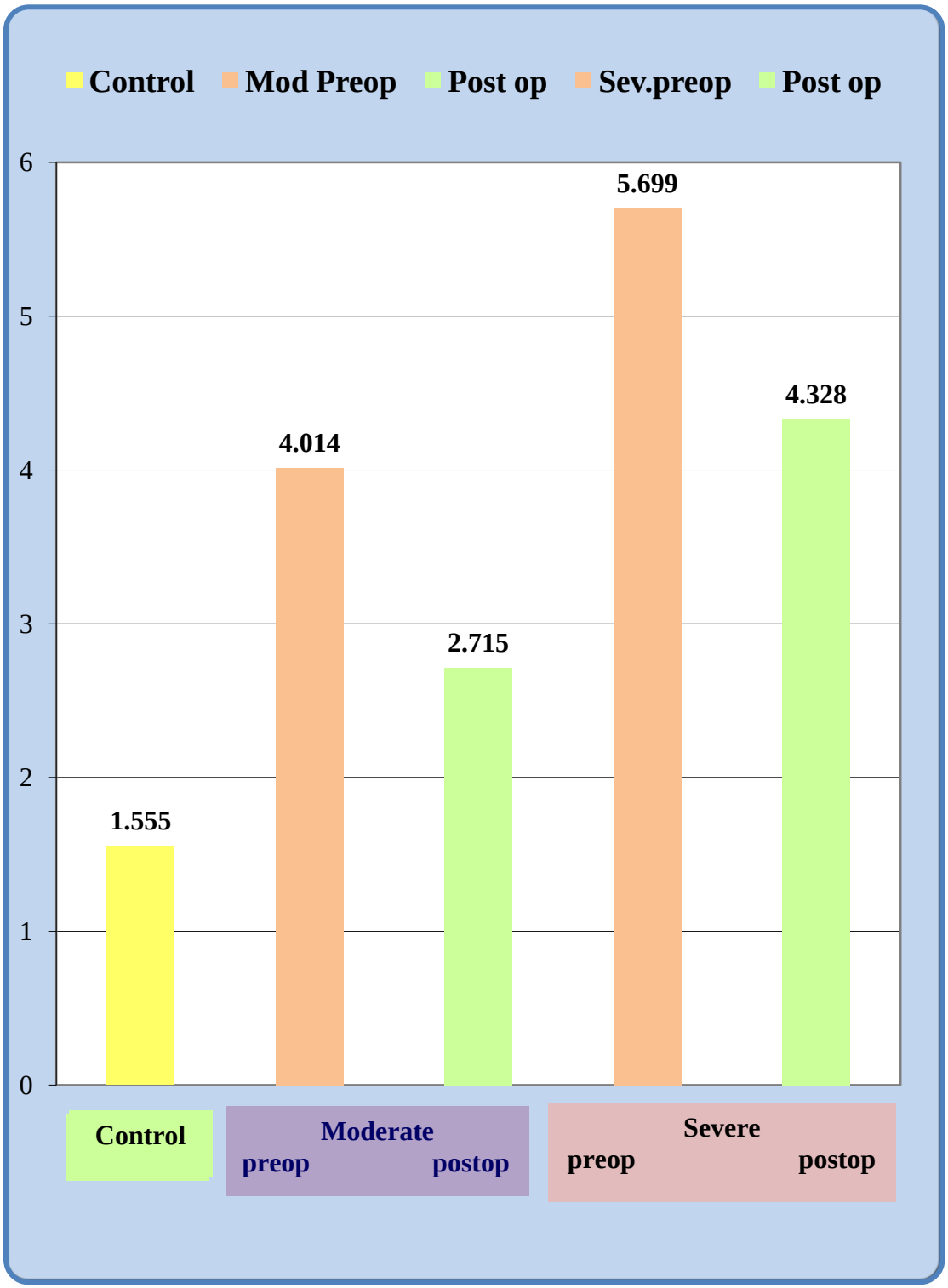


TABLE 6: COMPARISONS OF MEAN AND STANDARD DEVIATION (SD) OF HEMATOLOGICAL PARAMETERS AMONG CONTROL (GROUP I) AND TESTS GROUPS (GROUP II AND GROUP III) AT BASELINE AND I MONTH:

HEMATOLOGICAL PARAMETERS	BASELINE (MEAN±SD)				I MONTH (MEAN±SD)		
	Group I	Group II	Group III	p value	Group II	Group III	p value
LEUKOCYTE COUNT	7.25±2.556	8.447±1.878	8.48±1.286	0.09	7.191±1.822	7.39±1.9	0.737
NEUTROPHIL COUNT	4.02±2.263	5.394±1.496	5.24±1.09	0.024*	4.369±1.596	4.33±1.36	0.934
LYMPHOCYTE COUNT	2.06±0.538	2.446±0.555	2.59±0.57	0.011*	2.032±0.69	2.126±0.811	0.695
MONOCYTE COUNT	0.14±0.005	0.302±0.077	0.341±0.086	<0.001*	0.3±0.13	0.289±0.135	0.803
BASOPHIL COUNT	0.0535±0.015	0.015±0.005	0.0235±0.032	<0.001*	0.033±0.0729	0.0325±0.073	0.983
EOSINOPHIL COUNT	0.56±0.248	0.274±0.162	0.235±0.167	<0.001*	0.23±0.176	0.233±0.173	0.957
TOTAL RBC	5.092±1.83	4.881±0.882	5.01±0.67	0.862	6.437±1.318	5.46±0.71	0.06
Hb	9.362±1.26	7.745±0.868	8.449±1.225	<0.001*	8.834±2.216	9.19±1.02	0.518
MCV	100.485±6.322	93.28±11.143	95.785±7.611	0.033*	100.3±10.56	105.2±18.25	0.305
MCH	1.788±0.266	1.602±0.225	1.68±0.156	0.035*	1.628±0.213	1.694±0.17	0.286
MCHC	20.218±0.17	17.277±0.897	17.682±0.156	<0.001*	17.386±0.781	17.865±1.243	0.152
HEMOCRIT	0.372±0.051	0.437±0.032	0.434±0.0294	<0.001*	0.432±0.0371	0.434±0.0294	0.884
PLATELET COUNT	233.163±62.7	311.9±59.67	294.25±58.351	<0.001*	273.95±61.50	253.6±62.37	0.305
ESR	5.8±3.699	11.65±10.83	16.1±18.81	0.044*	6.7±5.695	6.06±9.05	0.79

*** -p value is significant**

The table 6 compared the mean and standard deviation value of leukocyte count in group I, group II and group III at baseline is 7.25±2.55, 8.447±1.878 and 8.48±1.286 respectively; neutrophil count in group I, group II and group III is 4.02±2.26, 5.39±1.49 and 5.24±1.09 respectively; lymphocyte count is 2.06±0.53, 2.45±0.55 and 2.59±0.57 respectively. The leukocyte counts among group I, group II and group III was not statistically significant. The neutrophil counts and lymphocyte counts among group I, group II and group III was statistically significant. .

The mean and standard deviation value of monocyte count at baseline in group I, group II and group III is 0.14 ±0.01, 0.302±0.07 and 0.341±0.08 respectively; basophil count at baseline is 0.05±0.02, 0.015±0.00 and 0.0235±0.03 respectively; eosinophil count is 0.56±0.25, 0.274±0.16 and 0.235±0.16 in group I, group II and group III respectively at baseline. There was statistically significant among the groups for monocyte counts, basophil counts and eosinophil counts (p value <0.001).

The mean and standard deviation of total RBC in group I, group II and group III was 5.09±1.83, 4.88±0.88 and 5.01±0.67 respectively at baseline. The mean and standard deviation of hemoglobin (Hb) level at baseline was 9.36±1.26, 7.745±0.86 and 8.449±1.22 respectively. There was significant difference among the groups for RBC count and Hb.

The mean and standard deviation of MCV is 100.49±6.32, 93.28±11.14 and 95.785±7.61 in group I, group II and group III respectively; MCHC is 20.22±0.17, 17.277±0.89 and 17.682±0.89 respectively; hemocrit value is 0.37±0.01, 0.437±0.032 and 0.434±0.029 respectively; platelet count is 233.16±62.7, 311.9±59.67 and 294.25±58.3 in group I,

group II and group III respectively at baseline. The difference among the values was statistically significant ($p<0.001$)

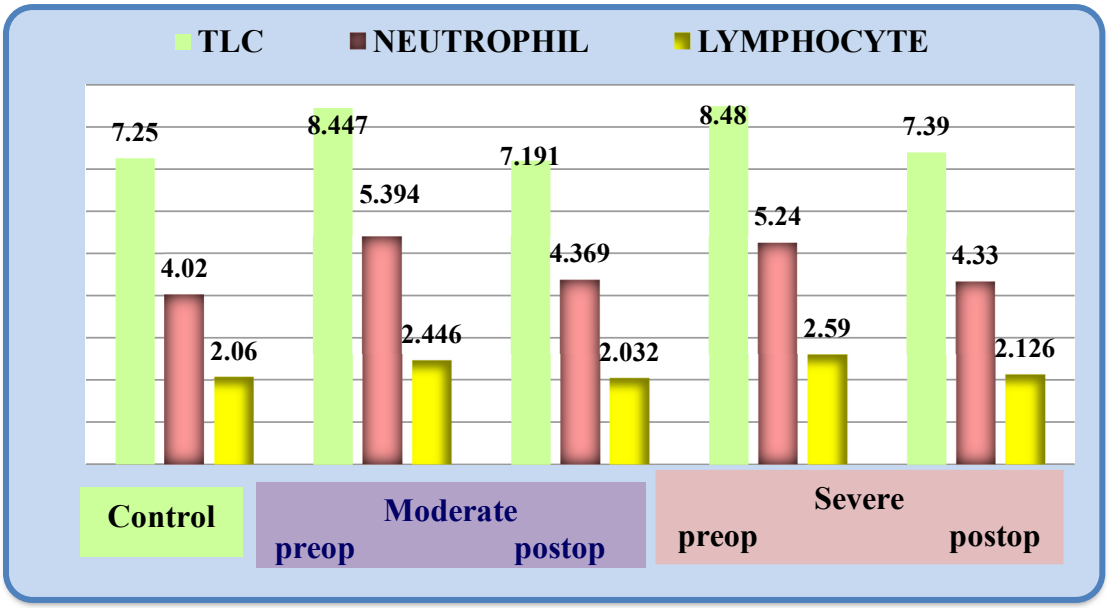
The mean and standard deviation of MCH values is 1.79±0.27, 1.602±0.22 and 1.68±0.156 in group I, group II and group III with satisfactory significant (p value 0.035) at baseline.

The mean and standard deviation of ESR at baseline is 5.8±3.69, 11.65±10.83 and 16.6±18.81 in group I, group II and group III respectively. The mean change in ESR value among the group showed satisfactory significant ($p= 0.04$).

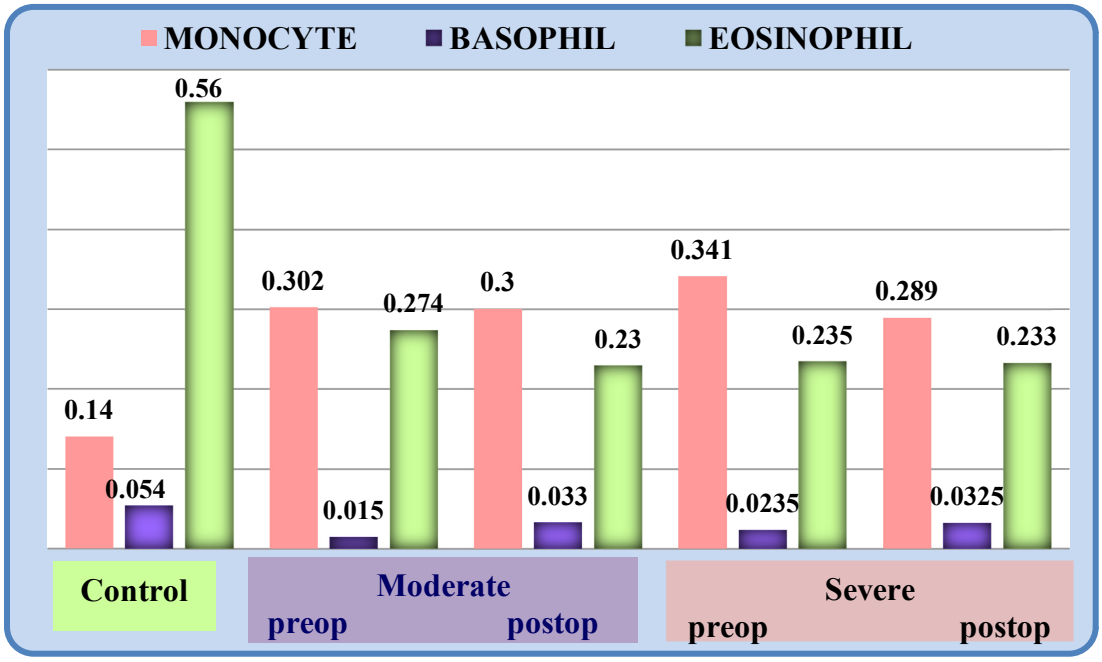
This table also showed the mean and standard deviation of hematological parameters after 1 month of nonsurgical periodontal therapy in group II and group III. The mean values for hematological parameters after 1 month was not statistically significant among group II and group III.

Graph 5 to 12 showed the graphical representation of hematological parameters in group I, group II and group III at baseline and I month.

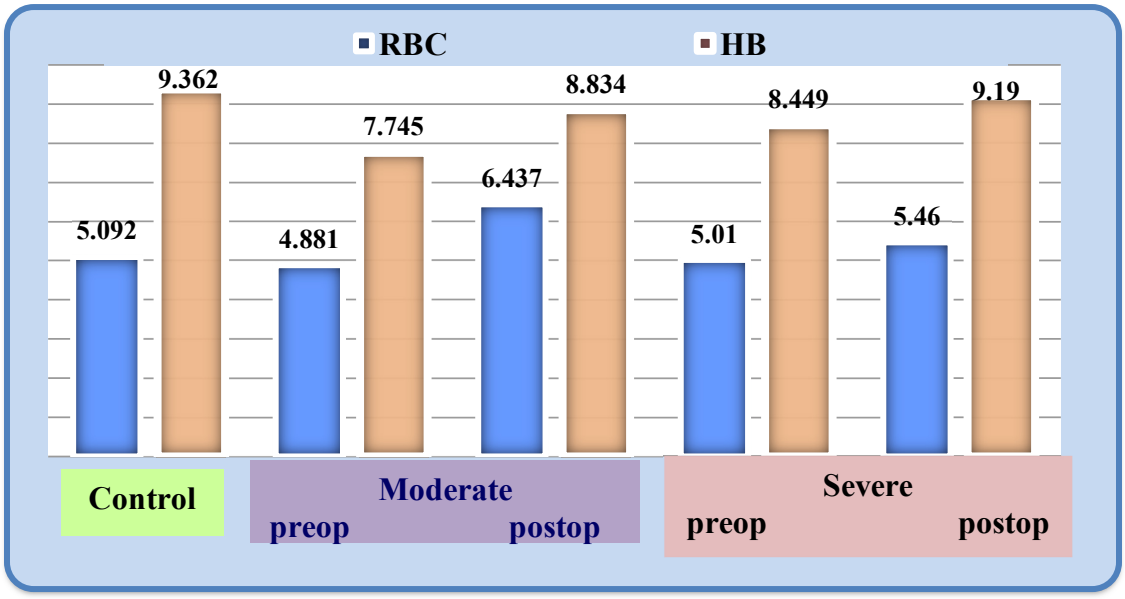
Graph 5: Comparison of leukocyte count, neutrophil count and lymphocyte count in group I, group II and group III at baseline and I month:



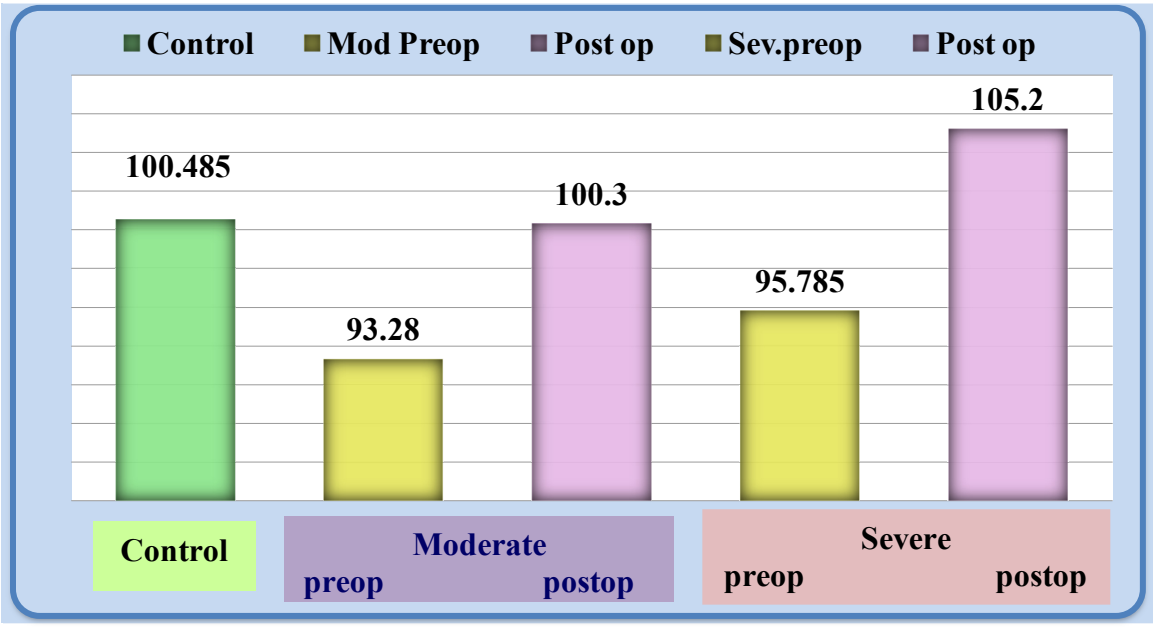
Graph 6: Comparison of monocyte count, basophil count and eosinophil count in group I, group II and group III at baseline and I month:



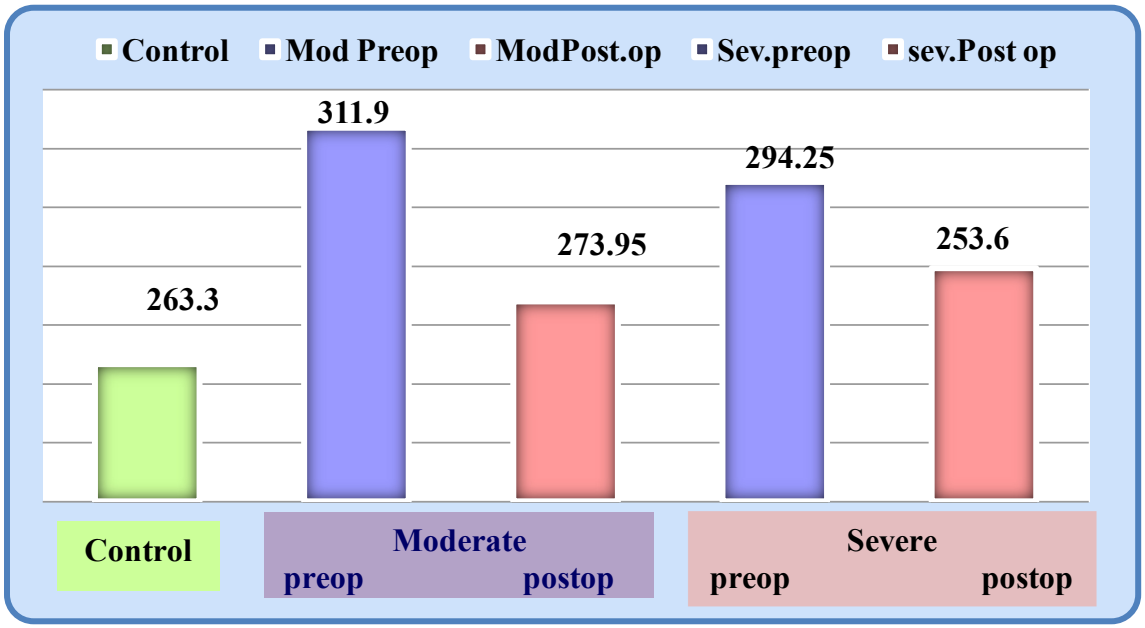
Graph 7: Comparison of RBC and Hb in group I, group II and group III at baseline and I month:



Graph 8: Comparison of MCV in group I, group II and group III at baseline and I month:



Graph 11: Comparison of platelet count in group I, group II and group III at baseline and I month



Graph 12: Comparison of ESR in group I, group II and group III at baseline and I month

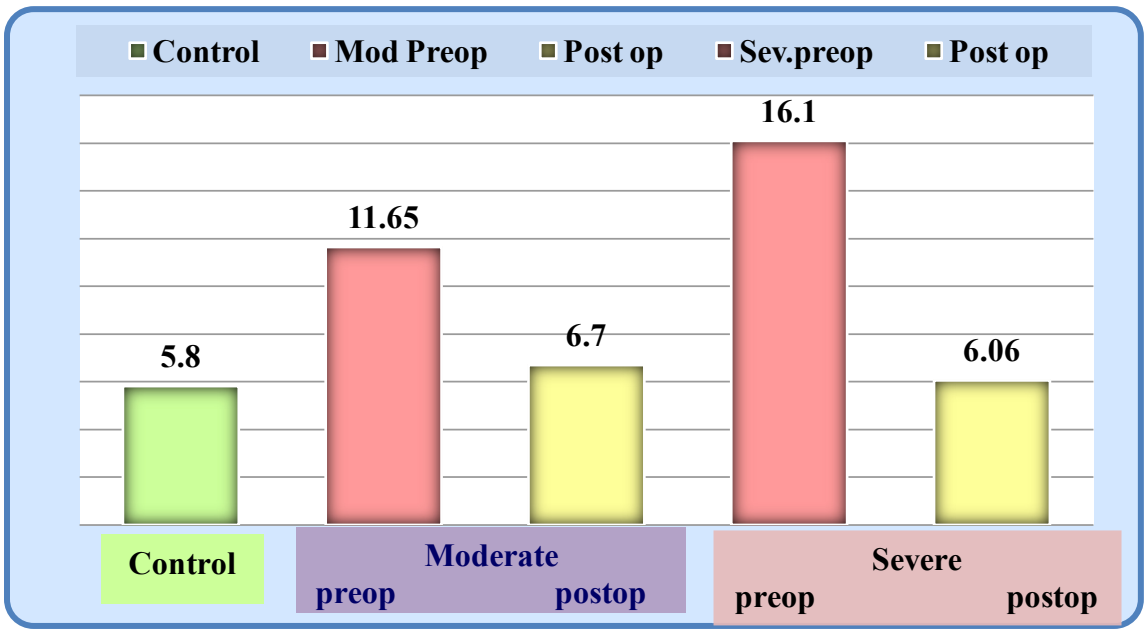


TABLE: 7 COMPARISONS OF MEAN AND STANDARD DEVIATION (SD) OF HEMATOLOGICAL PARAMETERS WITHIN GROUP II AND GROUP III AT BASELINE AND 1 MONTH:

HEMATOLOGICAL PARAMETERS	GROUP II (Mean±SD)			GROUP III (Mean±SD)		
	BASELINE	I MONTH	p VALUE	BASELINE	I MONTH	p VALUE
LEUKOCYTE COUNT	8.447±1.878	7.191±1.822	0.038*	8.48±1.286	7.39±1.90	0.029*
NEUTROPHIL COUNT	5.394±1.496	4.369±1.596	0.038*	5.24±1.09	4.33±1.36	0.028*
LYMPHOCYTE COUNT	2.446±0.555	2.032±0.69	0.044*	2.59±0.57	2.126±0.81	0.044*
MONOCYTE COUNT	0.302±0.077	0.3±0.13	0.948	0.341±0.086	0.289±0.13	0.618
BASOPHIL COUNT	0.015±0.005	0.033±0.0729	0.278	0.0235±0.032	0.0325±0.07	0.233
EOSINOPHIL COUNT	0.274±0.162	0.23±0.176	0.459	0.235±0.167	0.233±0.17	0.963
TOTAL RBC	4.881±0.882	6.437±1.318	0.05*	5.01±0.67	5.46±0.71	0.044*
Hb	7.745±0.868	8.834±2.216	0.048*	8.449±1.225	9.19±1.02	0.042*
MCV	93.28±11.143	100.3±10.56	0.047*	95.785±7.611	105.2±18.25	0.04*
MCH	1.602±0.225	1.628±0.213	0.174	1.68±0.156	1.694±0.17	0.784
MCHC	17.277±0.897	17.386±0.781	0.358	17.682±0.156	17.865±1.24	0.597
HEMOCRIT	0.437±0.032	0.432±0.0371	0.573	0.434±0.0294	0.434±0.02	0.979
PLATELET COUNT	311.9±59.67	273.95±61.50	0.049*	294.25±58.351	253.6±62.37	0.04*
ESR	11.65±10.83	6.7±5.695	0.043*	16.1±18.81	6.06±9.05	0.049*

*** -p value is significant**

DISCUSSION

DISCUSSION

Periodontitis is a multifactorial microbial disease initiated in the gingiva leading to bone destruction with the loss of other tooth supporting structures. The host responds to the periodontal infection with an array of cellular and molecular events involving both innate and adaptive immunity. Although periodontitis is a local infectious disease it also influences low grade systemic inflammation [**Ebersole and Cappelli 2000**].[36]

Chronic presence of periodontal disease leads to changes in systemic condition**.** In case of periodontal disease, it is speculated that inflamed and ulcerated subgingival pocket epithelium forms an easy port of entry for dental plaque bacteria with their dissemination of bacterial components into circulation. In response to bacteremia and dispersal of bacterial antigens, blood cellular changes produce pro–inflammatory mediators such as IL-1, IL-6, TNF-α, PGE_2 which exerts systemic effects.

The acute phase response is a non specific process that may occur in initial host response to injuries, infections, ischemic necrosis or malignancy. It is initiated by the activation of local macrophages and other cells leading to the release of mediators such as TNF-α, IL-6, IL-1β. An acute phase protein has been defined as the one whose plasma concentration increase (Positive acute phase proteins) or decreases (Negative acute phase proteins) by at least 25% during inflammatory disorders. These acute phase reactants have pro-inflammatory properties, activate complement factors, neutralize invasive pathogens and stimulate the repair and regeneration of variety of tissues.

The various acute phase proteins include CRP, transport proteins (haptoglobin, ceruloplasmin, and gamma 1-trypsin inhibitor), coagulation proteins (fibrinogen,

prothrombin) and complement components (C3, C4). Among the acute phase protein**,** C-reactive protein (CRP) is considered as a key marker of choice in monitoring the acute phase response because the markers tend to increase in higher concentration compared to basal concentration and is currently regarded marker of inflammation.

CRP is a plasma protein, pentameric in nature, synthesized by hepatocytes that participate in systemic response to inflammation. **Tillet and Francis [1930]** [123] discovered the presence of CRP in the serum of patients with the pneumonia because of its activity to react with C-polysaccharide isolated from pneumococcal cell walls. It is a pattern recognition molecule that binds to specific molecular configuration formed on the surface of pathogen [**Black et al 2004**].[15] It is regulated by interleukin-6, IL-1β and TNF-α. CRP levels guides decision regarding diagnosis, monitoring and therapy for various inflammatory reactions and linked disease [**Tillet et al]**.[123] It has extremely high sensitivity but very poor specificity as it is generated in response to many forms of injury.

For many years blood has been regarded as the ultimate body fluid that could indicate disease process. The blood cells have a vital role in supplying oxygen, maintaining hemostasis and providing protection to the periodontal tissues [**Iqbal et al 2015**].[56] In the blood, PMNs form the majority of white blood cells (40% to 72%), while lymphocytes range between 21% - 48%. Leukocytes are an integral part of innate immune system and these cells are recruited at higher levels during episodes of bacteremia in periodontitis or leakage in to systemic circulation **[Loos et al 2005].**[77] They are the major systemic cell of phagocytosis and first line of defence mechanism against infective agents. There were differences in the counts of the cells in various severities of periodontitis, due to accumulation of oral biofilm when compared to healthy controls. It has been reported in

several studies that number of leukocytes were elevated in periodontal disease compared to healthy subjects.

Lowered hemocrit values and hemoglobin levels have been reported in periodontitis patients due to chronic nature of disease. **Cartwright et al [1966]** [21] stated there were 3 factors related in decreasing the amount of hemocrit and erythrocytes: 1.reduced the survival of erythrocytes. 2. Bone marrow disorder reduces the amount of red blood cell. 3. Damage and defect of iron releasing from the reticuloendothelial system. The determination of ESR is helpful in curing patients with chronic inflammatory disorders. Assessing the platelet count is important because it has its main function in hemostasis and role in inflammatory and immune process. Increase in platelet count occurs in chronic inflammation and it can possibly explain the link between periodontal inflammation and CVD.

Chronic periodontitis is a disease characterized by loss of supporting tissue of the teeth, bone loss, mobility and exfoliation of the teeth. Early diagnosis is important in this disease which often remains unrecognizable until the onset of serious symptoms by **Ramachandra et al [2017]**.[104] Chronic periodontitis is known to have systemic effect due to its inflammatory nature. It has been shown that periodontal bacteria or their by products directly invade the periodontal tissue and gain access to the systemic circulation. Changes in the blood have been detected in patients with periodontitis. More commonly an elevation in the number of peripheral leukocytes and variation in the levels of serum proteins identified as acute phase protein was considered as a characteristic of infectious condition.

The treatment modalities for controlling the disease process by non surgical methods include scaling and root planing the root surface and motivating the patient to maintain oral health and control the risk factors. Nonsurgical periodontal treatment is a cornerstone of periodontal therapy usually first recommended approach to control periodontal infections. This therapy has evolved over the years and still considered to be the gold standard to which other treatment modalities are compared.

So evaluating the level of systemic cellular and molecular marker of inflammation will explain the association between periodontal disease and inflammatory burden. Systemic inflammation modifies the periodontitis principally by acting on the normal immune and inflammatory defence cells. **The present study evaluates the levels of systemic inflammatory markers such as WBC count, neutrophil count, lymphocyte count, platelet count and ESR along with the assessment of hs-CRP in this study because they reflect changes associated with the systemic inflammation.**

In our study we have included patient in age group of 25-65 yrs and elevation in the CRP levels in the test group >40 yrs would be due to changes in the lifestyle and presence of disease which is subclinical [**Kinane et al 2005].**[66] There were no significant differences observed among the sexes. Even though the CRP levels were slightly higher in woman, the difference was not stastically significant. However it is reported in many studies that there is a certain trend for CRP levels to be higher among women than men [**De Maat et al 2001].**[33]

Studies reporting association between destructive periodontal disease and CRP has varied results. The discrepancy in the study result may be due to the difference in the severity of

the periodontal disease and study population.[54] In the past CRP assays were not accurate and sensitive, so high sensitive CRP assays were used to detect CRP levels as low as 0.15 mg/L.

Recent evidence has indicated patients with severe periodontitis have increased serum levels of CRP when compared to the control group [**Gomes Filho et al 2011]** [46] which was comparable to our study results. Various studies have proved a positive association between presence of chronic periodontitis and high serum CRP levels [**Slade et al 2000** [117]**, Noack et al 2001** [89]**, Ebersole et al 2000** [36]] explaining the biological plausibility that inflammatory mediators (IL-1, IL-6, TNF-α) are released under conditions of periodontitis. This stimulates the hepatocytes to produce CRP thus explaining the increase in CRP levels in chronic periodontitis.

Elevated CRP is well recognized risk factor for the atherosclerotic complications upregulated by pro-inflammatory cytokines released locally at the site of inflammation **[Ridker et al 1998].**[106] Acute phase response was precipitated by severe or progressive destruction of periodontal disease that may provide possible link between two disease processes. Furthermore, CRP receptors on macrophages and neutrophil, targets the bacteria for phagocytosis and in turn damaged the host cells and amplify the response to infection.[15]

Kweider et al [1993] [70] reported higher numbers of leukocytes in periodontitis patients similar to our findings. A significant increase in neutrophil and lymphocyte counts was observed in periodontitis patients. **Siegel et al [1945]**[116] in his study showed the prevalence of secondary anemia in patients with periodontitis. A study by **Rahul et al**

[2013] [102] showed that number of erythrocytes and hemocrit levels were low in patients with periodontitis similar to the study done by **Latha et al [2015**] [71] in which anemia was more prevalent. **Khan et al [2014]** [64] in his study reported that there was significant reduction in the red blood parameters in accordance with our study results. In a similar study done by **Thomas et al [2006]**[122] showed that patients with periodontitis had lower level of erythrocyte, hemocrit and hemoglobin supported by the studies done by **Agarwal et al [2009]** [1] and **Gokhale et al [2010].**[45] **Al-Rasheed [2011]** [4] showed significant increase in WBC count and platelet counts in chronic periodontitis patients compared to the healthy control group, which supported the results of our study.

Despite the fact that poor periodontal health is linked to higher systemic CRP levels and the influence of successful periodontal therapy on CRP levels remain elusive. Following successful periodontal therapy, the bacterial load is significantly reduced and there is an improvement in the antibody titers to the specific pathogens. As the result of these changes there is a significant improvement in the clinical parameter as observed in our study.

Recent trial has indicated that treatment of periodontal disease either by intensive mechanical therapy, drug therapy or extraction can significantly lower serum levels of CRP. Decrease in the serum CRP was significantly associated with the periodontal treatment with the decrease in infection burden and the periodontal inflammation as assessed by clinical parameters [**Ioannidou et al 2006].**[55] **Ebersole et al [1997]** [37] was the first to investigate the effect of periodontal treatment on serum CRP levels wherein he used NSAID (flurbiprofen) in different doses on serum CRP levels. But the study results were not clearly conclusive regarding the pre and post treatment.

D'Aiuto et al [2005] [27] in his study reported a decrease in CRP levels after the completion of periodontal therapy which is comparable to our study results. In contrast to the results of the above study, **Ide et al [2003]** [53] in his study failed to observe a reduction in circulating CRP following treatment and the possible explanation given by the author was SRP is insufficient to control periodontal disease progression in all periodontitis subjects. Most of the periodontal intervention studies performed had excluded patients with a known propensity for atherosclerosis similar to our study. The rise in CRP levels in the presence of periodontal disease, and the potential reduction in the levels with treatment were greater in these patients.

Many studies **Siddeshappa et al [2016]** [115] **and Taylor et al [2010]** [120] reported that there was statistically significant decrease in total leukocyte count from baseline and after periodontal therapy which was similar to our study results. In our study there was statistically significant reduction in neutrophil and lymphocyte counts but eosinophil and monocyte differences observed in the test groups was not stastically significant. The study result was similar to the **Banthia et al [2013]**.[12]

Siegel et al [1945] [116] in his study showed improvement in hemoglobin and hemocrit values after SRP. Our study results showed improvement in Hb levels after SRP in accordance with the study done by **A.R.Pradeep et al [2011].**[100] Our results found to be consistent with the study by **Malhotra et al [2012]** [79]**,** where no significant changes observed after non surgical periodontal therapy in MCH, MCHC and hemocrit levels. This result was contrary to the study **by Parihar et al [2018]** [93]**,** where they stress significant improvement in all erythrocyte parameters.

Platelet count showed significant decrease after SRP similar to the results reported by **Christan et al [2002].**[24] In our study there was stastical significant decrease in ESR after SRP which is in accordance with study by **Hutter et al [2001].**[51] The positive effect of nonsurgical therapy in reducing the levels of cellular and molecular markers of inflammation could be helpful in preventing the risk of cardiovascular diseases.

Till date none of the studies have assessed CRP with all the hematological markers and their counts in different levels of periodontal severity and in 1 month interval. The ideal time for re-evaluation would be 4-8 weeks as agreed by the consensus report from AAP as there is an ongoing repair process during the above period. [111]

There are very few studies reported in literature which had a follow up interval of 1 month as in our study. Recalling the patients after 2 months may be long enough for the pathogenic bacteria to repopulate the periodontal pockets.

The strength of the present study was use of high sensitive immunonephlometry technique to evaluate CRP levels that was capable of detecting variations ≤0.2 mg/L. In the previous studies by **Ide et al [2003]** [53]**, Yamazki et al [2005]** [133], variety of techniques have been used along with the enzyme linked immunosorbent assay test and this creates difficulty in making comparisons between them.

Analysis of data from our study confirms the observations from previous studies that in case of otherwise healthy subjects with chronic periodontitis showed a moderate increase in systemic inflammation. Evaluation of systemic marker of inflammation by serological and hematological parameters serves as predictor of the future CVS disease **[Koeing et al 2000].**[67]

Although there is large body of evidence to indicate that serum CRP levels and leukocyte count are elevated in patients with chronic periodontal disease, further investigation is needed to support periodontal treatment intervention as a measure of systemic inflammation in the general population. It is highly unlikely that CRP levels and blood parameters can be modulated by single visit of non surgical treatment in patients with severe periodontal diseases.

More randomized controlled trials evaluating additional treatment modalities such as repeated scaling and root planing or surgical intervention are needed to accomplish a definitive and stable end point of treatment to demonstrate that CRP levels and blood counts can be affected by periodontal treatment.

There were some limitations in our study which includes smaller sample size, single course of treatment and short term follow up. Further longitudinal long term follows up and interventional studies are needed to explore the association between periodontitis and systemic disease and in particular the possible influence of periodontal therapy on the reduction of inflammatory markers and decrease risk of systemic disease.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

From the present study it can be summarized that

1. Chronic periodontitis results in **higher systemic levels** of CRP, white blood cell count, platelet count, ESR and a **decrease in all erythrocyte parameters** both moderate and severe groups.

2. There was **significant decrease** in the hs-CRP levels, total leukocyte count, neutrophil count, lymphocyte count, platelet count and ESR in otherwise healthy individuals affected with moderate and severe chronic periodontitis **after non surgical periodontal treatment. Improvement** in erythrocyte parameters was also observed **after non surgical periodontal treatment.**

3. Elevation in inflammatory mediators may **increase the risk of CVS events.** Thus chronic periodontitis may **add** to the inflammatory burden of individuals, by rising CRP levels and blood parameters thus increasing the risk of CVS disease. s

Based on the above fact it can be conclude that there is **decrease in a hs-CRP levels and hematological parameters after nonsurgical periodontal treatment**, which may reduce the risk of future CVS events thereby altering the systemic inflammation.

BIBLIOGRAPHY

1. Agarwal N, Kumar VS, Gujjari SA. Effect of periodontal therapy on hemoglobin and erythrocyte levels in chronic generalized periodontitis patients: An interventional study. J Indian Soc Periodontol 2009; 13(1):6-11.

2. Al Ghamdi AS. Effect of chronic periodontal diseases on erythrocyte sedimentation rate. Egypt Dent J 2009; 55:2641- 2648.

3. Aljohani HA. Association between haemoglobin level and severity of chronic periodontitis. JKAU Med Sci 2010; 17:53-60.

4. Al-Rasheed A. Elevation of white blood cells and platelet counts in patients having chronic periodontitis. Saudi Dental Journal 2012; 24: 17-21.

5. American Academy of Periodontology Task Force Report on the Update to the 1999 Classification of Periodontal Diseases and Conditions. J Periodontol 2015; 86(7):835-8.

6. Ana, Pejcic, et al. The Markers of Systemic Inflammation in Patients with Chronic Periodontitis: Leukocytes, C - reactive protein and Fibrinogen. J Prev Med 2013; 43-9.

7. Anitha G, Nagaraj M, Jayashree A. Comparative evaluation of levels of C-reactive protein and PMN in periodontitis patients related to cardiovascular disease. J Indian Soc Periodontol 2013; 17(3):330-32.

8. Annals of Periodontology, 1996 World Workshop in Periodontics. Published by the AAP 1996; 1:1.

9. Anumolu VN, Srikanth A, Paidi K. Evaluation of the relation between anemia and periodontitis by estimation of blood parameters: A cross-sectional study. J Indian Soc Periodontol 2016; 20(3):265-72.

10. Aziz AD, Kalekar M, Suryakar DN, Kale R , Benjamin T, Dikshit M. Pre- and Post-Treatment Effectiveness of SRP on Levels of IL-6, IL-10, and CRP in Chronic Periodontitis Patients with and without Diabetes. American Journal of Biochemistry 2018; 8(1):1-6.

11. Bansal T, Dhruvakumar D, Pandey A. Comparative evaluation of C-reactive protein in peripheral blood of patients with healthy gingiva, gingivitis and chronic periodontitis: A clinical and particle-enhanced turbidimetric immuno-analysis. J Indian Soc Periodontol 2014; 18(6):739-43.

12. Banthia R, Jain P, Banthia P, Belludi S, Parwani S, Jain A. Effect of phase I periodontal therapy on pro-coagulant state in chronic periodontitis patients – A clinical and hematological study. J Ir Dent Assoc 2013; 59:183-8.

13. Barot & chandran. Statistical Study on Serum CRP levels in Aggressive & Chronic Periodontitis: Pre & Post Non-Surgical Periodontal Therapy. Anaplastology 2015, 4:3.

14. Beck DJ, Offenbacher S. Relationships among clinical measures of periodontal disease and their association with systemic markers. Ann Periodontol 2002; 7(1): 79-89.

15. Black S, Kushner I, Samols D. C-reactive protein. J Biol Chem 2004; 279:48487–90.

16. Bokhari SA, Khan AA, Tatakis DN, Azhar M, Hanif M, Izhar M .Non-surgical periodontal therapy lowers serum inflammatory markers: a pilot study. J Periodontol 2009; 80:1574–80.
17. Bolla V, Kumari PS, Munnangi SR, Kumar DS, Durgabai Y, Koppolu P. Evaluation of Serum C-reactive Protein Levels in Subjects with Aggressive and Chronic Periodontitis in Comparison with Healthy Controls: A Clinico-biochemical Study. Int J Appl Basic Med Res 2017; 7(2):121-4.
18. Bretz WA, Weyant RJ, Corby PM, Ren D, Weissfeld L, Kritchevsky SB, et al. Systemic inflammatory markers, periodontal diseases, and periodontal infections in an elderly population. J Am Geriatr Soc 2005; 53:1532-7.
19. Buhlin K, Gustafsson A, Pockley AG, Frostegård J, Klinge B. Risk factors for cardiovascular disease in patients with periodontitis. Eur Heart J 2003; 24:2099-107.
20. Carranza, Newman, Takei and Klokkevold. Carranza's Clinical Periodontology, 12th Edition, 2015. Elsevier, Saunders.
21. Cartwright GE. The anemia of chronic disorders. Semin Hematol 1966; 3(4):351–75.
22. Chandy S, Joseph K, Sankaranarayanan A, Issac A, Babu G, Wilson B, et al. Evaluation of C-reactive protein and fibrinogen in patients with chronic and aggressive periodontitis: a clinico-biochemical study. J Clin Diagn Res 2017; 11(3):41-5.
23. Chandy S, Krishnan V, Anila S, Issac AV**.** Evaluation of peripheral blood levels in patients with chronic and aggressive periodontitis: A clinico- biochemical study. International Journal of Periodontology and Implantology 2018; 3(1):17-23.

24. Christan C, Dietrich T, Hagewald S, Kage A, Bernimoulin JP. White blood cell count in generalized aggressive periodontitis after non-surgical therapy. J Clin Periodontol 2002; 29:201-6.

25. Correa F. O, Goncalves D, Figueredo CM, Bastos AS, Gustafsson, A and Orrico SR. Effect of periodontal treatment on metabolic control, systemic inflammation and cytokines in patients with type 2 diabetes .Journal of Clinical Periodontology 2010; 37, 53–58.

26. Craig RG, Yip JK, So MK, Boylan RJ, Socransky SS, Haffajee AD. Relationship of destructive periodontal disease to the acute-phase response. J Periodontol 2003; 74:1007-16.

27. D'Aiuto F, Nibali L, Parkar M, Suvan J, Tonetti MS. Short-term effects of intensive periodontal therapy on serum inflammatory markers and cholesterol. J Dent Res 2005; 84:269-73.

28. D'Aiuto F, Parkar M, Andreou G, Brett PM, Ready D, Tonetti MS. Periodontitis and atherogenesis: causal association or simple coincidence? J Clin Periodontol 2004; 31:402-11.

29. D'Aiuto F, Parkar M, Nibali L, Suvan J, Lessem J, Tonetti MS. Periodontal infections cause changes in traditional and novel cardiovascular risk factors: results from a randomized controlled clinical trial. Am Heart J 2006; 151:977-84.

30. D'Aiuto F, Parkar M, Tonetti MS. Periodontal therapy: a novel acute inflammatory model. Inflamm Res 2005; 54(10):412-4.

31. D'Aiuto F, Ready D, Tonetti MS. Periodontal disease and C-reactive protein associated cardiovascular risk. J Periodontal Res 2004; 39:236-41.

32. D'Aiuto F, Graziani F, Tetè S, Gabriele M, Tonetti MS. Periodontitis: from local infection to systemic diseases. Int J Immunopathol Pharmacol 2005; 18(3):1-11.

33. De Maat MP, Kluft C. Determinants of C-reactive protein concentration in blood. Ital Heart J 2001; 2:189-95.

34. De Souza AB, Okawa RP, Silva CO, Araújo MG. Short-term changes on C-reactive protein (CRP) levels after non-surgical periodontal treatment in systemically healthy individuals. Clin Oral Investig 2017; 21(1): 477

35. Deepa D, Gupta C, Gupta A. Assessment of high-sensitivity C-reactive protein values in chronic periodontitis patients with and without cardiovascular disease: A cross-sectional study. J Clin Prev Cardiol 2016; 5:108-12.

36. Ebersole JL, Cappelli D. Acute-phase reactants in infections and inflammatory diseases. Periodontol 2000 2000; 23:19-49.

37. Ebersole JL, Machen RL, Steffen MJ, Willman DE. Systemic acute-phase reactants, C-reactive protein and haptoglobin in adult periodontitis. Clin Exp Immunol 1997; 107:347-52.

38. Elter JR, Hinderliter AL, Offenbacher S et al. The effects of periodontal therapy on vascular endothelial function: a pilot trial. Am Heart J 2006; 151:47.

39. Fredriksson M, Figueredo C, Gustafsson A, Bergstrom K, A˚ sman B. Effect of periodontitis and smoking on blood leukocytes and acute-phase proteins. J Periodontol 1999; 70:1355-60.

40. Freitas CO et al. Effect of non-surgical periodontal therapy on the levels of C-reactive protein: a pilot study. Rev Odonto Cienc 2011; 26(1):16-21.

41. Gani DK, Lakshmi D, Krishnan R, Emmadi P. Evaluation of C-reactive protein and Interleukin-6 in peripheral blood of patients with chronic periodontitis. J Indian Soc Periodontol. 2009; 13(2):69-74.

42. Gani DK, Mallineni SK, Ambalavanan, Ramakrishnan, Deepalakshmi, Emmadi P. Estimation of the levels of C-reactive protein, interleukin-6, total leukocyte count, and differential count in peripheral blood smear of patients with chronic periodontitis in a South Indian population. West Indian med j 2012; 61(8):826-31.

43. George AK, Janam P. The short-term effects of non-surgical periodontal therapy on the circulating levels of interleukin-6 and C-reactive protein in patients with chronic periodontitis. J Indian Soc Periodontol 2013; 17(1):36-41.

44. Glurich I, Grossi S, Albini B, et al. Systemic inflammation in cardiovascular and periodontal disease: Comparative study. Clin Diagn Lab Immunol 2002; 9:425-32.

45. Gokhale SR, Sumanth S, Padhye AM. Evaluation of blood parameters in patients with chronic periodontitis for signs of anemia. J Periodontol 2010; 81:1202-6.

46. Gomes-Filho IS, Freitas Coelho JM, da Cruz SS, Passos JS, Teixeira de Freitas CO, Aragao Farias NS, et al. Chronic periodontitis and C-reactive protein levels. J Periodontol 2011; 82:969-78.

47. Graziani F, Cei S, Tonetti M, Paolantonio M, Serio R, Sammartino G, Gabriele M and D'Aiuto F. Systemic inflammation following non-surgical and surgical periodontal therapy. J Clin Periodontol 2010; 37(9):848-54.

48. Hada, DS, Garg S, Ramteke GB and Ratre MS. Effect of Non-Surgical Periodontal Treatment on Clinical and Biochemical Risk Markers of Cardiovascular Disease: A Randomized Trial. J Periodontol 2015; 86: 1201-11.

49. Haghoo JM, Gholami L, Tahepour O, Ebrahimi S. The evaluation of the effects of nonsurgical periodontal treatment on blood parameters of hemocrit, RBCS, hemoglobin, CRP and LDL/VLDL among patients with chronic periodontitis. Annals of dental speciality 2018; 6(2):132-6.

50. Hegde S, Riyas, Kashyap R, Kumar AMS. Comparative evaluation of blood parameters in chronic periodontitis patients and healthy patients for signs of anemia. Journal of dental and medical sciences 2014; 13(12)30-6.

51. Hutter JW, van der Velden U, Varoufaki A, Huffels RA, Hoek FJ, Loos BG. Lower numbers of erythrocytes and lower levels of hemoglobin in periodontitis patients compared to control subjects. J Clin Periodontol 2001; 28:930-6.

52. Ide M, Jagdev D, Coward PY, Crook M, Barclay GR, Wilson RF. The short-term effects of treatment of chronic periodontitis on circulating levels of endotoxin, C-reactive protein, tumor necrosis factor-alpha, and interleukin-6. J Periodontol 2004; 75:420-8.

53. Ide M, McPartlin D, Coward PY, Crook M, Lumb P and Wilson RF. Effect of treatment of chronic periodontitis on levels of serum markers of acute-phase inflammatory and vascular responses. J Clin Periodontol 2003; 30:334–40.

54. Imaki M, Ogawa Y, Yoshida Y, Uchida M, Tanada S. Cohort study of the total leukocyte count and periodontal disease among company employees. Environmental Health and Preventive Medicine 1999; 4:54-7.

55. Ioannidou E, Malekzadeh T, Dongari-Bagtzoglou A. Effect of periodontal treatment on serum C-reactive protein levels: A systematic review and meta-analysis. J Periodontol 2006; 77:1635-42.

56. Iqbal PS, Khan SN, Haris M, Narayanan M, Laju S, Kumar SS. Assessment of Systemic Inflammatory Markers in Patients with Aggressive Periodontitis. J Int Oral Health 2015; 7(Suppl 2):48-51.

57. Jain K, Das SJ, Dwivedi S, Jain R, Nugala B and Jain M. Effect of periodontal treatment on red blood cell parameters in patients with chronic periodontitis. IJDRD 2016, 6(3):13-8.

58. Jayaprakash D, Aghanashini S, Chatterjee A, Bharwani A, Vijayendra R, Rosh R. Effect of periodontal therapy on C-reactive protein levels in gingival crevicular fluid of patients with gingivitis and chronic periodontitis: a clinical and biochemical study. J Indian Soc Periodontol 2014; 18(4):456–60.

59. Joshipura KJ, Wand HC, Merchant AT, Rimm EB. Periodontal disease and biomarkers related to cardiovascular disease. J Dent Res 2004; 83:151–5.

60. Kahn S, Imperial RC, Menezes CC, Dias AT, Rodrigues WJ, Barceleiro M and Moreira**.** Effects of non-surgical periodontal treatment on complete blood count, lipid and glycemic profiles of patients with an indication for surgical coronary revascularization. Rev Bras Odontol 2017; 74(3):203-9.

61. Kalsi DS, Sood A, Mundi S, Sharma V. Effect of scaling and root planing on blood counts in patients with chronic generalized periodontitis. Indian J Dent Sci 2017; 9:109–13.

62. Kamil W, Al Habashneh R, Khader Y, et al. Effects of nonsurgical periodontal therapy on C-reactive protein and serum lipids in Jordanian adults with advanced periodontitis. J Periodontal Res 2011; 46:616-21.
63. Kanaparthy A, Kanaparthy R, Niranjan N. Evaluation of serum C-reactive protein levels in subjects with aggressive and chronic periodontitis and comparison with healthy controls. Dent Res J 2012; 9:261-5.
64. Khan NS, Iqbal S, Haris M, Chandramohan S and Kumar S. Relationship between Hemoglobin Level and Severity of Chronic Periodontitis. Int J Chem & Life Sci 2014; 3(1):1269-73.
65. Khan NS, Luke R, Soman RR, Krishna PM, Safar IP, Swaminathan SK. Qualitative assessment of red blood cell parameters for signs of anemia in patients with chronic periodontitis. J Int Soc Prev Community Dent 2015; 5(6):476-81.
66. Kinane DF, Riggio MP, Walker KF, MacKenzie D and Shearer B Bacteraemia following periodontal procedures. J Clin Periodontol 2005; 32:708–13.
67. Koening W. Heart disease and the inflammatory response. BNJ 2000; 321:187-8.
68. Kolte RA, Kolte AP, Deshpande NM. Assessment and comparison of anemia of chronic disease in healthy subjects and chronic periodontitis patients: A clinical and hematological study. J Indian Soc Periodontol 2014; 18:183-6.
69. Kumar BP, Khaitan T, Ramaswamy P, Sreenivasulu P, Uday G, Velugubantla RG et al. Association of chronic periodontitis and white blood cell and platelet count- a case control study. Journal of Clinical and Experimental Dentistry. 2014; 6(3):214-17.

70. Kweider M, Lowe GD, Murray GD, Kinane DF, McGowan DA. Dental disease, fibrinogen and white cell count; links with myocardial infarction? Scott Med J 1993; 38:73-4.

71. Latha S, Thirugnanamsambandan S, Arun RT, Masthan KM, Malathi L, Rajesh E. Serum ferritin level and red blood cell parameters in healthy controls chronic periodontitis patients. J Pharm Bioallied Sci 2015; 7(Suppl 1): S184-9.

72. Leite AC, Carneiro VM, Guimaraes C. Effects of periodontal therapy on C-reactive protein and HDL in serum of subjects with periodontitis. Rev Bras Cir Cardiovasc 2014; 29:69-77.

73. Leivadaros E, Van der Velden U, Bizzarro S, et al. A pilot study into measurements of markers of atherosclerosis in periodontitis. J Periodontol 2005; 76:121- 28.

74. Linden GJ, McClean K, Young I, Evans A, Kee F. Persistently raised C-reactive protein levels are associated with advanced periodontal disease. J Clin Periodontol 2008; 35: 741–47.

75. Loe H. The gingival index, plaque index and the retention index systems. J Periodontol 1967; 38:610-616.

76. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PME, van der Velden U. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol 2000; 71:1528-34.

77. Loos BG. Systemic markers of inflammation in periodontitis. J Periodontol 2005; 76: 2106-15.

78. Lopez R, Loos BG, Baelum V. Hematological features in adolescents with periodontitis. Clin Oral Investig 2012; 16:1209–16.

79. Malhotra R, Kapoor A, Grover V, Grover D and Kaur A. Effect of scaling and root planning on erythrocyte count, hemoglobin and hematocrit in patients with chronic periodontal disease. J Dent Hyg 2012; 86(3):195-203.

80. Mallapragada S, Kasana J, Agrawal P. Effect of nonsurgical periodontal therapy on serum highly sensitive capsule reactive protein and homocysteine levels in chronic periodontitis: A pilot study. Contemp Clin Dent 2017; 8:279-85.

81. Marcaccini AM, Meschiari CA, Sorgi CA, Saraiva MCP, Souza AM, Faccioli LH, Tanus-Santos JE, Novaes AB, Gerlach RF. Circulating interleukin-6 and high-sensitivity C-reactive protein decrease after periodontal therapy in otherwise healthy subjects. J Periodontol 2009; 80:594–602.

82. Mattila K, Vesanen M, Valtonen V, Nieminem M, Palosuo T, Rasi V et al. Effect of treating periodontitis on C-reactive protein levels: a pilot study. BMC Infect Dis 2002; 2:30.

83. Mishra P, Agarwal S, Devraj CG, Nayak PA, Yadav A, Sharma S. Determination of Erythrocyte parameters in Chronic periodontitis patients. International journal of Medical Science and education 2014; 1(3):148-54.

84. Miyasaki KT. The neutrophil: Mechanisms of controlling periodontal bacteria. J Periodontol 1991; 62:761-74.

85. Montebugnoli L, Servidio D, Miaton RA, Prati C, Tricoci P, Melloni C, et al. Periodontal health improves systemic inflammatory and haemostatic status in subjects with coronary heart disease. J Clin Periodontol 2005; 32:188-92.

86. Monteiro AM, Jardini MA, Alves S, Giampaoli V, Aubin EC, Neto AM, Gidlund M. Cardiovascular disease parameters in periodontitis. J. Periodontol 2009; 80(3):378–88.

87. Musalaiah SV, Anupama M, Nagasree M, Krishna CM, Kumar A, Kumar PM. Evaluation of nonsurgical periodontal therapy in chronic periodontitis patients with anemia by estimating hematological parameters and high-sensitivity C-reactive protein levels. J Pharm Bioallied Sci 2014; 6 (Suppl 1): S64-9.

88. Nakajima T, Honda T, Domon H, Okui T, Kajita K, Ito H et al. Periodontitis-associated up-regulation of systemic inflammatory mediator level may increase the risk of coronary heart disease. J Periodontal Research 2010; 45:116–22.

89. Noack B, Genco RJ, Trevisan M, Grossi S, Zambon JJ, De Nardin E. Periodontal infections contribute to elevated systemic C-reactive protein level. J Periodontol 2001; 72:1221-7.

90. Offenbacher S, Madianos PN, Champagne CM. Periodontitis-atherosclerosis syndrome: an expanded model of pathogenesis. J Periodontol Res 1999; 34: 346-352.

91. Pabolu CM, Mutthineni RB, Chintala S, N, Mutthineni N. Evaluation of the effect of one stage versus two stage full mouth disinfection on C-reactive protein and leucocyte count in patients with chronic periodontitis. J Indian Soc Periodontol 2013; 17:466-71.

92. Paraskevas S, Huizinga JD, Loos BG. A systematic review and meta-analyses on C-reactive protein in relation to periodontitis. J Clin Periodontol 2008; 35:277-90.

93. Parihar S, Kumar N, Bhatnagar A, Parihar AV. Evaluating the Effect of Periodontal Therapy on Hematological Parameters among Subjects with Chronic Periodontitis: An Interventional Study. J Oral Health Comm Dent 2018; 12(1):25-30.

94. Patel MD, Shakir QJ, Shetty A. Interrelationship between chronic periodontitis and anemia: A 6-month follow-up study. J Indian Soc Periodontol 2014; 18(1):19-25.

95. Patil VA, Desai MH. Effect of periodontal therapy on serum C-reactive protein levels in patients with gingivitis and chronic periodontitis: a clinico-biochemical study. J Contemp Dent Pract 2013; 14(2):233-7.

96. Pejcic A, Kesic L, Pesic Z, Mirkovic D, Stojanovic M. White blood cell count in different stages of chronic periodontitis. Acta Clin Croat 2011; 50(2):159-67.

97. Pejcic A, Kesic LJ, Milasin J. C-reactive protein as systemic marker of inflammation in periodontitis. Eur J Clin Microbiol Infect Dis 2011; 30(3): 407-414.

98. Persson GR, Pettersson T, Ohlsson O, Renvert S. High-sensitivity serum C-reactive protein levels in subjects with or without myocardial infarction or periodontitis. J Clin Periodontol 2005; 32:219-24.

99. Podzimek S, Mysak J, Janatova T, Duskova J. C-Reactive Protein in Peripheral Blood of Patients with Chronic and Aggressive Periodontitis, Gingivitis, and Gingival Recessions. Mediators Inflamm 2015; ID:564858.

100. Pradeep AR, Anuj S. Anemia of chronic disease and chronic periodontitis: does periodontal therapy have an effect on anemic status? J Periodontol 2011; 82(3):388-94.

101. Prakash S, Dhingra K, Priya S. Similar hematological and biochemical parameters among periodontitis and control group subjects. European Journal of Dentistry 2012; 6:287-94.

102. Rahul P. Evaluation of hematological changes in patients with chronic periodontitis & gingivitis in comparison to healthy controls - A clinical study. J Dent Allied Sci 2013; 2(2):49-53.

103. Rai B, Kaur J, Kharb S, Jain R, Anand SC, Singh J et al. Peripheral blood and CRP levels in chronic periodontitis. African journal of Biochemistry research 2009; 3(4):150- 153.

104. Ramachandra SS, Gupta VV, Mehta DS, Gundavarapu KC, Luigi N. Differential diagnosis between chronic versus aggressive periodontitis and staging of aggressive periodontitis: A cross-sectional study. Contemp Clin Dent 2017; 8:594-603.

105. Ramich T, Asendorf A, Nickles K, Oremek GM, Schubert R, Nibali L, Wohlfeil M, Eickholz P. Inflammatory serum markers up to 5 years after comprehensive periodontal therapy of aggressive and chronic periodontitis. Clin oral investigations 2018; 27:1-1.

106. Ridker PM, Cushman M, Stampfer JM, Tracy PR, Hennekens HC. Plasma concentration of C-reactive protein and risk of developing peripheral vascular disease. Circulation 1998; 97:425–48.

107. Russell AL. The Periodontal Index. J Pedodontol 1967; 38:586-91.

108. Saito T, Murakami M, Shimazaki Y, Oobayashi K, Matsumoto S, Koga T. Association between alveolar bone loss and elevated serum C-reactive protein in Japanese men. J Periodontol 2003; 74:1741-1746.

109. Sambashivaiah S, Rebentish PD, Kulal R, Bilchodmath S. One stage vs. two stage Non-surgical Periodontal therapy and their effect on WBC count. Int. Journal of Clinical Dental Science 2010; 1.

110. Sangeetha S et al. Effect of non surgical periodontal therapy on hematological parameters in chronic periodontitis patients. JIDENT 2017; 1(3):1-6.

111. Segelnick SL, Weinberg MA. Reevaluation of initial therapy: When is the appropriate time? J Periodontol 2006; 77:1598-601.

112. Seinost G, Wimmer G, Slerget M, et al. Periodontal treatment improves endothelial dysfunction in patients with severe periodontitis. Am Heart J 2005; 149:1050-4.

113. Shimada Y, Komatsu Y, Ikezawa-Suzuki I, Tai H, Sugita N, Yoshie H. The effect of periodontal treatment on serum leptin, interleukin-6, and C-reactive protein. J Periodontol 2010; 81:1118-23.

114. Shojaee M, Fereydooni Golpasha M, Maliji G, Bijani A, Mir SM, Kani SN. Reactive Protein Levels in Patients with Periodontal Disease and Normal Subjects. Int J Mol Cell Med 2013; 2(3):151– 5.

115. Siddeshappa ST, Nagdeve S, Yeltiwar RK, Parvez H, Deonani S, Diwan V. Evaluation of various hematological parameters in patients with periodontitis after nonsurgical therapy at different intervals. J Indian Soc Periodontol 2016; 20:180-3.

116. Siegel EH. Total erythrocyte, leucocyte and differential white cell counts of blood in chronic periodontal disease. Journal of Dental Research 1945; 24:270-1.

117. Slade GD, Offenbacher S, Beck JD, Heiss G. Acute-phase inflammatory response to periodontal disease in the US population. J Dent Res 2000; 79(1): 49-57.

118. Solomon S, Pasarin L, Ursarescu I, Martu I, Bogdan M, Nicolaiciuc O et al. The effect of non-surgical therapy on C reactive protein and IL-6 serum levels in patients with periodontal disease and atherosclerosis. Int J Clin Exp Med 2016; 9(2):4411-7.

119. Suchetha A, Chandran N, Bhat D, Sapna N, Sravani K and Mundinamane DB. Comparative evaluation of hematological parameters in periodontally healthy, gingivitis and periodontitis subjects. J. Chem. Pharm Res 2015, 7(8):407-13.

120. Taylor BA, Tofler GH, Carey HMR, Morel-Kopp MC, Philcox S, Carter TR. Full-mouth Tooth Extraction Lowers Systemic Inflammatory and Thrombotic Markers of Cardiovascular Risk. Journal of Dental Research 2006; 85(1), 74–8.

121. Thakare KS, Deo V, Bhongade ML. Evaluation of the C-reactive protein serum levels in periodontitis patients with or without atherosclerosis. Indian J Dent Res 2010; 21:326-9.

122. Thomas B, Ramesh A, Rithesh K. Relationship between periodontitis and erythrocyte count. J Indian Soc Periodontol 2006; 10:288–91.

123. Tillett WS, Francis T. Serological reactions in pneumonia with a non-protein somatic fraction of pneumococcus. J Exp Med 1930; 52:561-71.

124. Tonetti MS, D'Aiuto F, Nibali L, Donald A, Storry C, Parkar, M, et al. Treatment of periodontitis and endothelial function. N Engl J Med. 2007; 356:911-20.

125. Tuter G, Kurtis B, Serdar M. Evaluation of gingival crevicular fluid and serum levels of high sensitivity C-reactive protein in chronic periodontitis patients with or without coronary artery disease. J Periodontol 2007; 78:2319-24.

126. Ushida Y, Koshy G, Kawashima Y et al. Changes in serum interleukin-6, C-reactive protein and thrombomodulin levels under periodontal ultrasonic debridement. J Clin Periodontol 2008; 35:969–75.

127. Vaishali S, Praveen S, Vishunupriya V and Gayathri R. WBC Count in Patients with Chronic Periodontitis. Int J Pharm Sci Rev Res 2016; 39(2):16-9.

128. Wakai K, Kawamura T, Umemura O, et al. Associations of medical status and physical fitness with periodontal disease. J Clin Periodontol 1999; 26:664- 72.

129. Wang X, Han X, Guo X, Luo X, Wang D. The effect of periodontal treatment on hemoglobin A1C levels of diabetic patients: a systematic review and meta-analysis. PLOS One 2014; 9: 108412

130. Wilson RF. Effect of treatment of chronic periodontitis on levels of serum markers of acute-phase inflammatory and vascular responses. J Clin Periodontol 2003; 30:334-40.

131. Wu T, Trevisan M, Genco RJ, Falkner KL, Dorn JP, Sempos CT. Examination of the relation between periodontal health status and cardiovascular risk factors: Serum total and high density lipoprotein cholesterol, C-reactive protein, and plasma fibrinogen. Am J Epidemiol 2000; 151:273-82.

132. Yamamoto T, Tsuneishi M, Furuta M, Ekuni D, Morita M, Hirata Y. Relationship between decrease of erythrocyte count and progression of periodontal disease in a rural Japanese population. J Periodontol 2011; 82(1):106-13.

133. Yamazaki K, Honda T, Oda T, Ueki-Maruyama K, Nakajima T, Yoshie H et al. Effect of periodontal treatment on the C-reactive protein and proinflammatory cytokine levels in Japanese periodontitis patients. J Periodontal Res 2005; 40:53-8.

134. Zhou SY, Duan XQ, Hu R, Ouyang XY. Effect of non-surgical periodontal therapy on serum levels of TNF-a, IL-6 and C-reactive protein in periodontitis subjects with stable coronary heart disease. Chin J Dent Res 2013; 16(2):145-51.

ANNEXURES

ANNEXURES

The Diocese of Madurai - Ramnad
C.S.I. College of Dental Sciences and Research
129, East Veli Street, Madurai - 625 001. Tamilnadu, India.
Ph : 0452 - 2321708, 2336604 Fax : 2336605
Email ID : csidental@gmail.com Website : www.csidentalcollege.org

ETHICAL COMMITTEE

Title of the work : Comparative evaluation of Serum CRP levels and complete blood count in Chronic Periodontitis Patients Before and After Nonsurgical Periodontal Therapy.

Principal investigator :Dr.G.Kalaivani.

Department: Dept of Periodontics **CSICDSR/IEC/0027/2016**

The request for an approval from the Institutional Ethical Committee (IEC) for the above mentioned study, submitted by the principal investigator is considered on the IEC meeting held on 08.09.2016 at CSI College of Dental Sciences and Research, Madurai. The members of the committee, the president, vice president, and the secretary are pleased to approve the proposed work mentioned above and is '**Advised to proceed with the study**'

The principal investigator and their team are directed to adhere the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate from the area of work for which you have applied for ethical clearance.
5. You should inform the IEC immediately in case of any adverse events or serious adverse reactions. You should abide to the rules and regulations of the institution(s).
6. You should complete the work within the specific period and if any extension of time is required, you should apply for the permission again and do the work.
7. You should submit the summary of the work to the ethical committee on completion of the work.
8. You are advised to standardizethe study with IOPA, OPG and proceed further.
9. You should understand that the members of IEC have the right to monitor the work with prior intimation.
10. Your work should be carried out under the direct supervision of your Guide/Professor.

Dr.A.Charles MS MCh
President

Dr.N.Gururaj MDS
Secretary

INFORMATION SHEET

We are conducting a study on **COMPARATIVE EVALUATION OF SERUM C-REACTIVE PROTEIN AND COMPLETE BLOOD COUNT IN CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER NONSURGICAL PERIODONTAL THERAPY.** The identity of the patients participating in the research will be kept confidential throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared. Taking part in the study is voluntary. You are free to decide whether to participate in the study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled. The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Name of the patient Signature / Thumb impression

Name of the investigator: **Signature**

Date:

INFORMED CONSENT

Name: Age/Sex: O.P.no:

Address:

I, ———————— age ——— years exercising my free power of choice, hereby give my consent to be included as a participant in the study "comparative evaluation of serum c - reactive protein and complete blood count in chronic periodontitis patients before and after nonsurgical periodontal therapy. ***I agree to the following:***

1. I have been informed to my satisfaction about the purpose of the study and study procedures including investigations to monitor and safeguard my body function.
2. I agree to co-operate fully for complete examination.
3. I agree to report to my doctor for regular follow ups, when required for the research.
4. I hereby give permission to use my medical records for research purpose. I am told that the investigating doctor and institution will keep my identity confidential.
5. I understand that I have rights to withdraw myself from the study and also that the investigator has the right to exclude me from the research at any point of time. I am willing to give my blood as a sample for research.

Name of the patient Signature / Thumb impression

Name of the investigator Signature

ஆராய்ச்சி ஒப்புதல் படிவம்

சி.எஸ்.ஐ பல்மருத்துவக்கல்லூரி மற்றும் ஆராய்ச்சி மையம் , மதுரை.

ஈறுநோயால் பாதிக்கப்படும் நோயாளிகளுக்கு ஈறு சிகிச்சை மூலம் இரத்தத்தில் உள்ள CRP மற்றும் முழு இரத்த அணுக்கள் எண்ணிக்கையை (CBC) முன்பும், பின்பும் மதிப்பிட்டு அதனை ஒப்பிட்டு ஆராய்ச்சி செய்தல்.

பெயர்: தேதி:

வயது: புறநோயாளியின் எண்:

பாலினம்: ஆராய்ச்சி செயற்கை எண்:

கீழ்காணப்படும் நிபந்தனைகளுக்கு நான் ஒப்புதல் அளிக்கிறேன்

· இந்த ஆராய்ச்சியின் நோக்கமும், செயல்முறைகளும் எனக்கு திருப்தியளிக்கும் வகையில் அறிவுறுத்தப்பட்டது.

· 4 மில்லி லிட்டர் இரத்தம் பரிசோதனைக்கு இரண்டு முறை எடுக்கப்படும் என்றும் பற்பரிசோதனைக்குப் பின்பு பல் சுத்தம் செய்யப்படும் என்றும் தெரிவிக்கப்பட்டது.

· ஆராய்ச்சிக்கான எனது உடல்நிலை, இதற்கு முன்பும், பின்பும், தற்போழுது எடுத்துக்கொள்ளும் சிகிச்சை பற்றியும் பல் மருத்துவர் கு.கலைவாணி (ஈறுநோய் அறுவைசிகிச்சை பிரிவு) அவரால் எனக்கு விரிவாகவும், தமிழில் எளிமையாகவும் எடுத்துரைக்கப்பட்டது. அதிலுள்ள சந்தேகங்கள், பக்கவிளைவுகள் பற்றி கேட்கவும் எனக்கு வாய்ப்பளிக்கப்பட்டது.

· என் மருத்துவகுறிப்பேடுகளை இந்த ஆராய்ச்சியில் பயன்படுத்திக் கொள்ள சம்மதிக்கிறேன். இந்த ஆராய்ச்சி மையமும், ஆராய்ச்சியாளரும் என்னுடைய விவரங்கள் அனைத்தையும் ரகசியமாக வைப்பதாக அறிகிறேன்.

_____ _____ _____

நோயாளியின் பெயர் கையொப்பம் தேதி

_____ _____ _____

ஆராய்ச்சியாளரியின் பெயர் கையொப்பம் தேதி

CASE PROFORMA

COMPARATIVE EVALUATION OF SERUM C-REACTIVE PROTEIN AND COMPLETE BLOOD COUNT IN CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER NONSURGICAL PERIODONTAL THERAPY

Name : **age /sex:**

Op no:

Address:

Phone no:

Chief complaints:

History of present illness:

Past dental history:

Past medical history:

Habits:

Oral hygiene measures:

Clinical examination:

Intra oral examination:

Hard tissues:

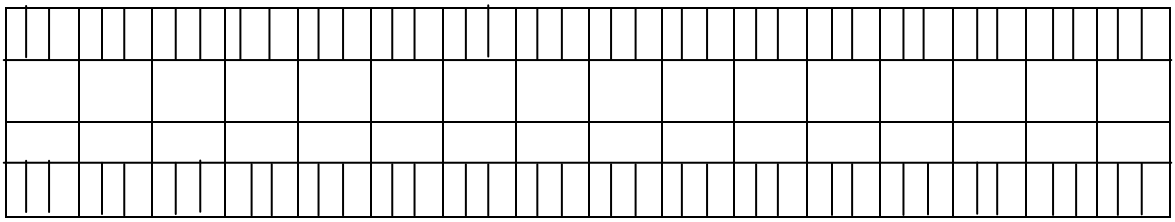
Soft tissues:

Gingiva:

PLAQUE INDEX - SILNESS AND LOE (1964):

At baseline:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

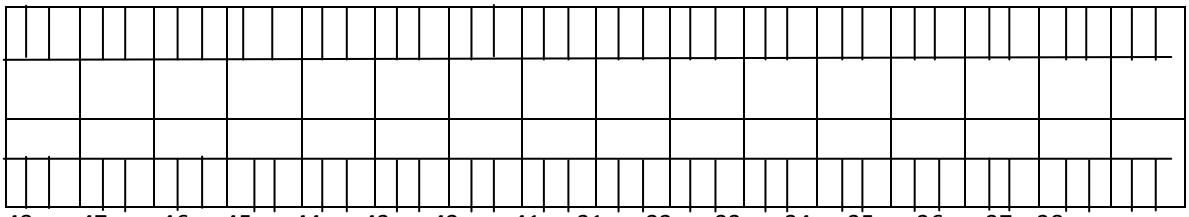


48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

PI score =

After one month

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28



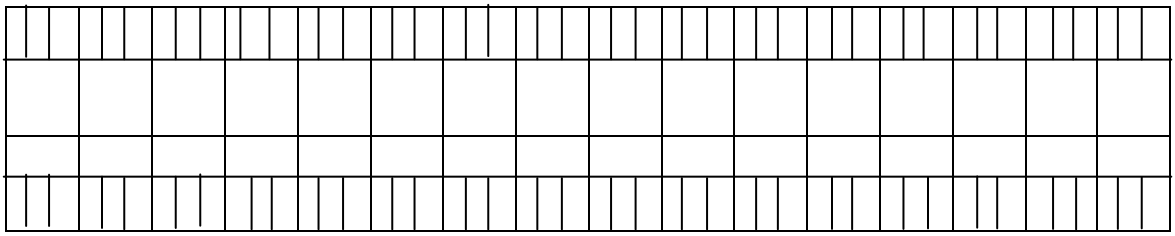
48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

PI score =

GINGIVAL INDEX - LOE AND SILNESS (1963):

At baseline:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

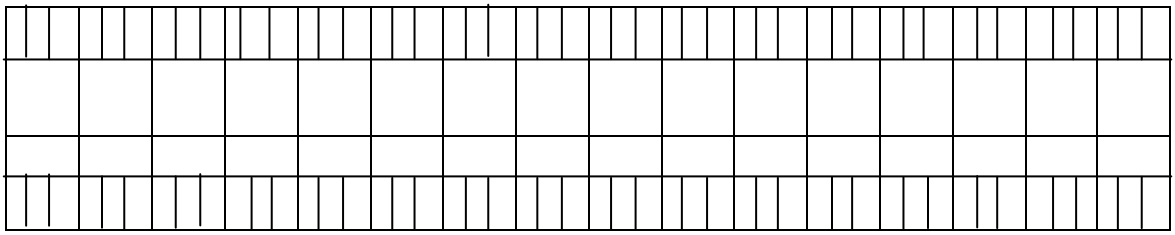


48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

GI score =

After one month:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28



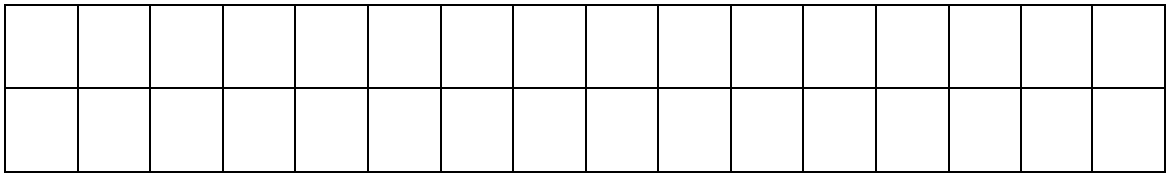
48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

GI score =

PAPILLARY BLEEDING INDEX - MUHLEMANN (1977):

At baseline:

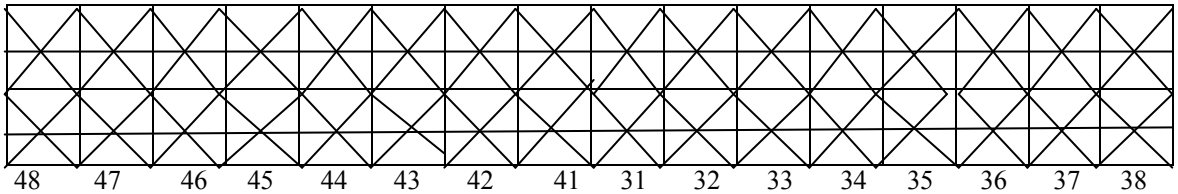
18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28



48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

After one month:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

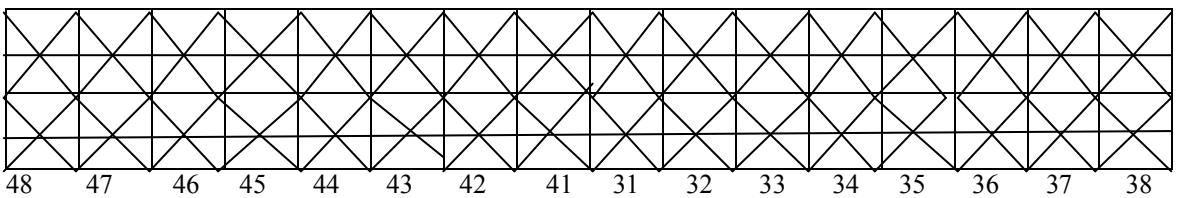


48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

PERIODONTAL PROBING DEPTH:

At baseline:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

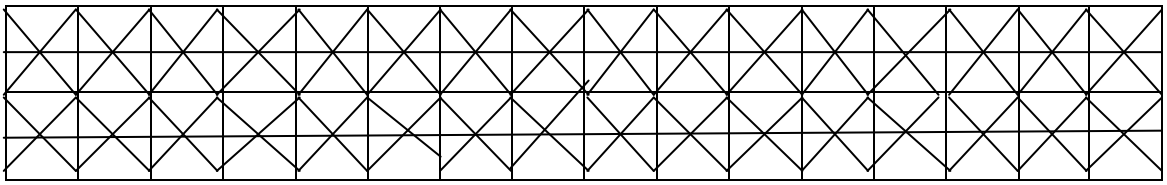


48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

PPD score =

After one month:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28



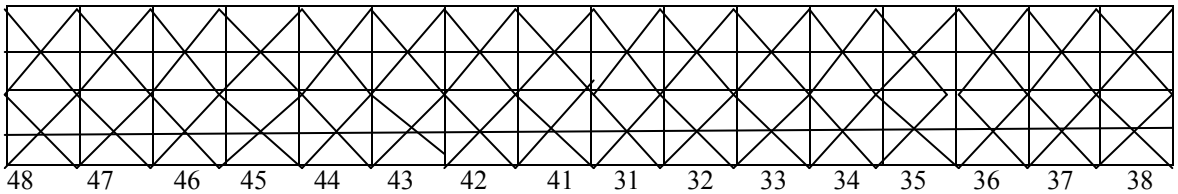
48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

PPD score =

CLINICAL ATTACHMENT LEVEL:

At baseline:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

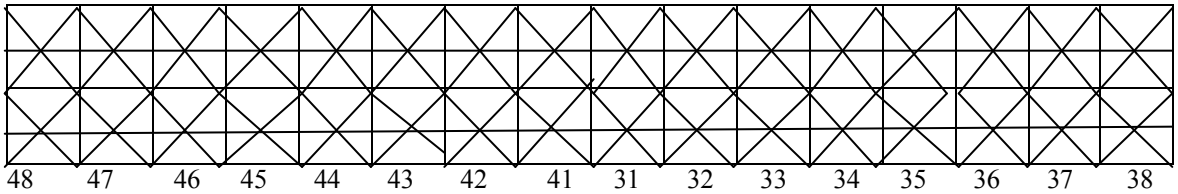


48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

CAL score =

After one month:

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28



48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

CAL score =

MOBILITY:

FURCATION:

DIAGNOSIS:

INVESTIGATION:

hs C-REACTIVE PROTEIN LEVEL:

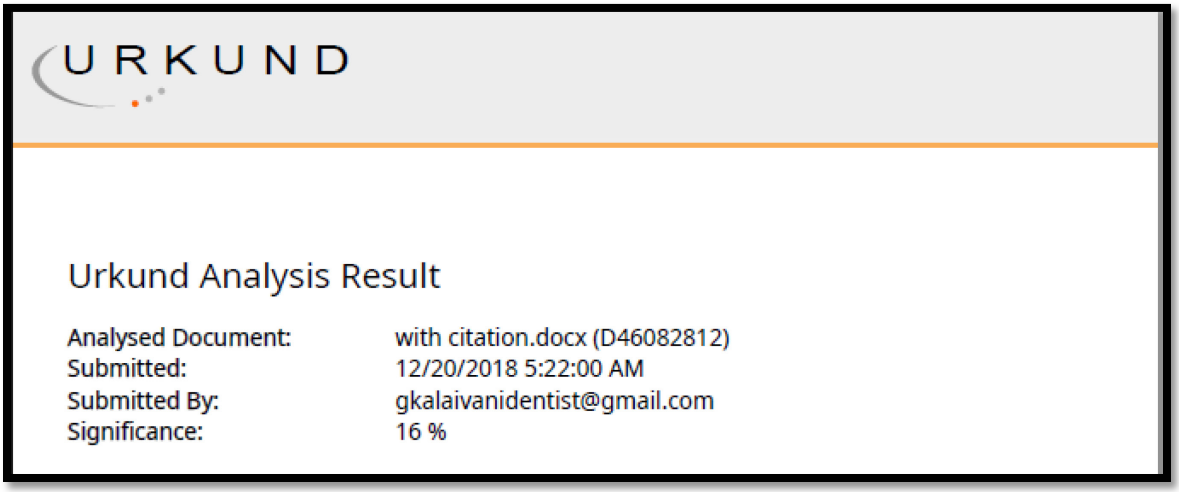
Baseline	After 1 month

COMPLETE BLOOD COUNT:

Tests	Baseline	After 1 month
Total leukocytes count		
Neutrophil count		
Lymphocyte count		
Eosinophil count		
Monocytes count		
Basophil count		
Hb		
RBC count		
Platelet count		
ESR		

Treatment plan: For group II and group III

Scaling	Root planing	OHI instructions	Recall visit After 1 month


URKUND
Urkund Analysis Result
Analysed Document: with citation.docx (D46082812)
Submitted: 12/20/2018 5:22:00 AM
Submitted By: gkalaivanidentist@gmail.com
Significance: 16 %